Sketchy Pathology



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Updated January 2018





- 1. **Oxidative forge:** cardiac myocytes generate energy almost exclusively through oxidative phosphorylation
- Oxygen bellows: myocytes extract a higher percentage of oxygen from blood than any other tissue in the body → coronary sinus has most deoxygenated blood
- 3. **Dilated red exhaust pipe**: coronary endothelial cells produce NO, a gaseous molecule that promotes coronary vascular vasodilation
- GruMPy blacksmith: NO ↑ cyclic GMP inside arteriolar smooth muscle cells ↑ coronary vasodilation
- 5. **Dancing with dilated red sleeves**: adenosine is an important vasodilator of coronary arteries
- Flat autoregulation graph: autoregulation (via NO and adenosine) normally keeps the coronary blood flow constant across a range of BPs by regulation coronary vasodilation
- 7. **Gunky constricted exhaust pipe**: coronary atherosclerosis obstructs luminal flow AND inhibits endothelial cell release of NO and other vasodilation
- Broken autoregulation gauge: coronary atherosclerosis interferes with autoregulation mechanism and ability to maximally vasodilate
- 9. Glowing harp: well oxygenated myocardium
- 10. Cold inner harp surface: subendocardium becomes ischemic first
- 11. "Supply and demand": mismatch between myocardial oxygen demand and coronary oxygen supply → ischemic heart disease
- 12. **Pushing load (Princess pushing blacksmith)**: Increased afterload on the heart (e.g. due to aortic stenosis or HTN)
- 13. Stenotic aortic princess hat and high pressure steam: aortic stenosis and HTN ↑ afterload →↑myocardial o2 demand
- 14. Concentric conch shell: concentric myocardial hypertrophy (due to ↑ afterload) → ↓coronary O2 supply
- 15. Diamonds on left: the LEFT ventricle receives coronary blood flow during diastole
- 16. Raised heart watch and falling diamond: tachycardia ↓ time in diastole → coronary flow to LEFT ventricle → myocardial ischemia
- 17. **Running blacksmith**: exercise ↑ myocardial O2 demand (tachycardia and ↑contractility) and ↓ coronary O2 supply (tachycardia)
- 18. Little constricted coronary crown: cocaine causes coronary artery vasoconstriction → ↓coronary O2 supply
- Jittery cocoa mug: cocaine ↑ myocardial O2 demand (tachycardia and Increased contractility)
- Pale complexion: systemic hypoxia ↓ coronary O2 supply (e.g. hypotension, shock, anemia and carbon monoxide poisoning)

- 21. **3 falling P batteries**: within seconds, ischemic myocardial cells switch from aerobic to anaerobic glycolysis → depleted ATP
- 22. Floppy harp strings: myofibril relaxation seen within Seconds (depleted ATP in the cardiac myocyte)- lack of crossbridge formation = ↓Cardiac output
- 23. Puffy harp repairman with candy bar and ball of string: early pathologic changes in ischemic myocyte include cellular and mitochondrial swelling, glycogen depletion, and clumping of chromatin (REVERSIBLE cell damage)
- 24. **Repairing harp with new red string:** cellular swelling and other early changes are reversible with early reperfusion
- 25. "Repaired in 30 min or less": irreversible damage to cardiac myocyte after ~30 minutes to ischemia
- Ruptured and vacuolated mitochondrial lute: mitochondrial vacuolization or membrane rupture is a sign of IRREVERSIBLE cell injury
- 27. **Spilling ChicKen and T-bone steaks:** myocyte cell membrane breakdown (IRREVERSIBLE cell damage) → release of troponin and creatine kinase
- 28. Stunned girl receiving repaired harp: STUNNED myocardium (viable myocytes do not immediately return to full activity) can last a few hours to days after reperfusion
- Hypercontracted lute: buildup of intracellular Ca during ischemia → hypercontracture and cytoskeletal damage on reperfusion (REPERFUSION INJURY)
- 30. **Sparks and fire caused by repairman**: reperfusion results in local release of free radicals, influx of inflammation, and further irreversible mitochondrial damage (REPERFUSION INJURY)
- 31. **Old grandfather clock:** Chronic ischemic heart disease (aka "ischemic cardiomyopathy")→progressive heart failure
- 32. Patched-up and discarded lute frame: Chronically ischemic hearts usually have evidence of "patchy fibrosis" from previous healed infarcts (more rarely, chronic severe CAD w/o infarct can also cause chronic ischemic heart disease
- 33. Floppy heart balloon above diluted lute frame: Chronic cardiac ischemia→ systolic heart failure with eccentric hypertrophy
- 34. **Hibernating bear:** chronically ischemic hearts may contain nonviable myocardial cells along with "hibernating" viable myocardial cells
- 35. Waking hibernating bear with vascular hose: revascularize a chronically ischemic heart to reactive viable myocardial cells (reverse systolic dysfunction, e.g. stent, bypass graft)

15. Ruptured stable: plaque rupture

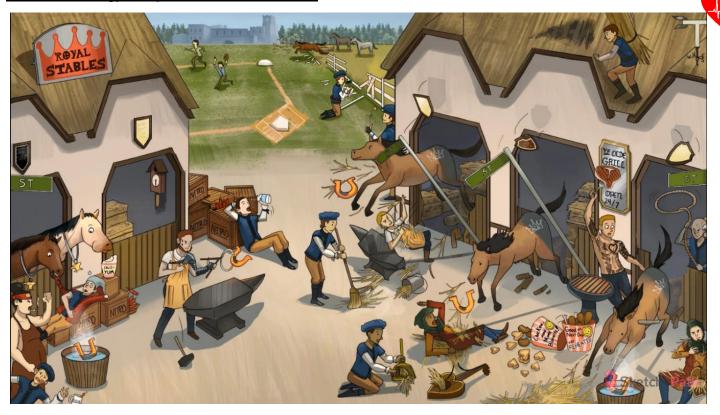
thrombus formation

16. Thrombotic hay released toward home plate: atheromatous plaque rupture lead to exposure of thrombogenic substances (ie. Tissue factor, Collagen) → platelet and coagulation pathway activation → luminal



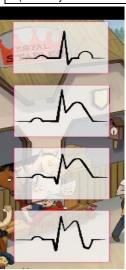


N. 7. 7. 44	
"70"-shaped tongs: fixed coronary plaques causing >70% stenosis will present clinically (stable angina)	17. Repairing ruptured fence: plaque rupture is quite common (repeated cycles of plaque disruption and repair are usually subclinical!)
Clutching chest over anvil: stable angina (predictable episodes chest pain and pressure worse with exertion) Plaques secured above stable: stable fixed atherosclerotic plaques	18. "ACS" horse rupture: acute coronary syndrome (ACS) includes unstable angina, NSTEMI, STEMI (destabilized coronary plaque → occlusive thrombus → cardiac ischemia or infarction)
cause stable angina (predictable symptoms) 4. Dark spot on inner horseshoe surface: stable angina involves	19. Disrupted plaque: ACS occurs due to acute plaque destabilization (rupture, erosion)
transient subendocardial ischemia 5. Tired blacksmith with armor on left arm: typical symptoms of stable angina include chest pressure/pain which can radiate to left arm and chin, diaphoresis and dyspnea (elderly diabetic, female pts may have minimal atypical sx)	20. Hay scattered from rupture: Plaque rupture/erosion exposes prothrombotic surface →occlusive luminal thrombus →cardiac ischemia or infarction (ACS)
	21. Flipping coin: we have no good way of predicting which plaques are vulnerable to rupture (NOT necessarily the largest or most occlusive)
 Relaxing on nitro crate: stable angina is relieved with rest or nitroglycerine (usually <30 min) 	22. Partially filled lumen: UNSTABLE ANGINA is caused by a partially occlusive (or transient) thrombus
7. Sleeping stable boy: vasospastic (Prinzmetal) angina symptoms occur at rest, more commonly at night	 Falling anvil: UNSTABLE ANGINA presents with unstable symptoms (new onset angina, angina with less exertion or at rest)
Twisted red sleeve: vasospastic angina is caused by transient coronary vasospasm	 Broken heart string: during NSTEMI, ischemia progresses to infarction and myocardial cell death
9. Startled awake: the paroxysmal vasospasm seen in vasospastic angina is likely due to smooth muscle hyper-reactivity in the coronary	 Disrupted plaque: ACS (e.g. Unstable angina) occurs due to acute plaque destabilization (rupture, erosion)
artery wall 10. Plaque mounted above stable: paroxysmal vasospasm seen in vasospastic (prinzmetal) angina common occurs over a stable atherosclerotic coronary plaque (may occur in disease free vessels)	26. Falling StreeT sign: NST"ACS" horse rupture through gate: Acute Coronary Syndrome (ACS) includes unstable angina, NSTEMI, STEMI (destabilized coronary plaque →occlusive thrombus → cardiac ischemia or infarction)
Relaxing on nitro box: nitroglycerin improves symptoms of vasospastic angina	27. ELEVATED StreeT sign: vasospastic angina presents with transient ST segment elevation in ECG leads that correspond to the region of
12. "Calci-Yum" icecream: calcium channel blockers are the 1st-line therapy for long term management of vasospastic (prinzmetal) angina (vasodilated and ↓spasticity)	ischemic myocardium 28. Sweeping up thrombotic hay: Unstable ANGINA is caused by a transient (or only partially occlusive) thrombus
13. Smoker : cigarette smoking is a major risk factor for prinzmetal angina (encourage pts to quite)	29. Dark spot on inner horseshoe surface: NSTEMI involves subendocardial region of infarction/cell death (ST depressions on
14. Sumo stable manager with cocoa kid: <u>sumatriptans</u> and sympathomimetic agents (e.g. cocaine, amph) can precipitate vasospastic angina	ECG = subendocardial involvement)
1	1



- 30. Completely filled lumen of lute: STEMI is caused by a fully occlusive (or prolonged) thrombus
- 31. Entire Thickness of horseshoe dark: STEMI angina involves transmural infarction/ cell death (ST elevation on ECG = transmural involvement)
- 32. T weather vane: hyperacute T waves within seconds corresponding to area of ischemia (STEMI progression of ECG changes 1of4)
- 33. Elevated ST: "tombstone" shaped ST elevation within minutes, corresponding to the area of infarction (STEMI progression of ECG changes 2of4)
- 34. "Q" lasso: after initial ST changes, negative Q waves develop corresponding to the area of necrosis (STEMI progression of ECG changes 3of4)
- 35. **Inverted T shadow:** after initial ST changes, **T wave inversion** (STEMI progression of ECG changes 4of4)
- 36. Pathologically old stable keeper: pathologic Q waves persists (area of previous infarct)
- 37. **Thatcher with bundle of stick(on the roof):** new -onset of LBBB is also diagnostic of STEMI (LBB is supplied by LAD)
- 38. Falling StreeT sign: unstable angina will present with ST segment depressions in ECG leads that correspond to the region of ischemia
- Dark spot on inner horseshoe surface: unstable angina involves sunendocardial ischemia (ST depressions on ECG = subendocardial involvement)
- 40. Entire Thickness of horseshoe dark: vasospastic (prinzmetal) angina involves transient transmural ischemia (ST elevation on ECG = transmural involvement)
- 41. ELEVATED StreeT sign: vasospastic angina presents with transient ST segment elevation in ECG leads that correspond to the region of ischemic myocardium

- 42. **Grillmaster holding T-Bone steak:** Death and rupture of cardiac myocytes in NSTEMI and STEMI leads to spillage of cellular enzymes, one of which is **Troponin**
- 43. **T-bone steak**: Troponins are serum markers used to detect infarction (dx NSTEMI n STEMI) (Troponin I is more SPECIFIC for cardiac muscle damage, troponin <u>T</u> is more SENSI<u>T</u>IVE)
- 44. CkicKen bucked "now with More Biscuits!": CK, isoform MB (**CKMB**) is a cardiac -specific serum marker used to detect infarction (dx NSTEMI and STEMI)
- 45. **T-bone 24/7**: serum troponin levels peak ~24hr after MI and remain ↑ for ~7 days
- 46. "Good the next day": serum CK-MB levels peak in ~24 hours and fall over the next ~24 hrs
- 47. **"try rehydrated**": serum CKMB can be used to dx **reinfarction** (relatively short time course of ↑ and return to baseline



Cardiac Pathology: Chapter 1.3 Acute Myocardial infarction and post-MI timeline



- 1. Broken heart string: myocardial infarction (MI)
- Crushing angina anvil: symptoms of MI include intense substernal chest pain or pressure that can radiate to the left arm or jaw
- Anxious jester: symptoms of MI include diaphoresis, anxiety, and dyspnea
- Discarded pills: symptoms of MI usually last >30min and are NOT relieved by rest or nitroglycerine
- Failing heart balloon on LEFT : MI (with LAD occlusion) can cause left sided heart failure (myocardial ischemia → decreased ventricular systolic function)
- 6. Left anterior braid: most MIs are caused by occlusion to the left anterior descending artery (LAD - branch of left coronary) → ischemia of anterior wall of the L ventricle, anterior ¾" of the interventricular septum, and apex
- 7. **Wet tunic with crackling bubbles on left**: MI with LAD occlusion can present with flash pulmonary edema and bibasilar crackles
- 8. Winded servant on left: MI with LAD occlusion can present with dyspnea
- Stiff S4 chair: MI can present with an S4 heart sound (ischemic heart tissue becomes stiff and noncompliant)
- Lightning sigil on left: MI with LAD occlusion can cause cardiogenic shock
- Dead jester with quivering heart: ventricular ischemia from MI can cause conduction abnormalities—fatal ventricular arrhythmias (e.g. V fib→ sudden cardiac death
- 12. Music notes on right: MI with occlusion of the right coronary artery (RCA) affects the SA and AV nodes (e.g. bradyarrhythmia, sick sinus syndrome)
- 13. Falling heart watch on right: MI with occlusion of the RCA can cause bradycardia (due to SA node dysfunction)
- 14. Heart shield on right: MI with occlusion of the RCA can cause heart block (due to AV node dysfunction)
- 15. **Right arm around back**: the RCA perfuses the R ventricle, posterior â..." of the interventricular septum and posterior L ventricle
- 16. Floppy heart balloon on right : MI with RCA occlusion can cause right sided heart failure (myocardial ischemia → decreased ventricular systolic function)
- Distended jug: MI with RCA occlusion can present with JVD (due to right heart failure)

- 18. Lightning sigil on right: MI with RCA occlusion can cause cardiogenic shock
- "II, III, aVF" triangle: MI with RCA occlusion can present with ST elevations on the inferior leads (II, III, aVF)
- 20. 4 clock hat: histopathology of MI 0-4 hrs
- 21. Normal fabric pattern : there are few histological changes 0-4 hrs post-
- 22. Half-sun mask: histopathology of MI 4-12 hrs (~half day)
- 23. Wavy tassels: "wavy fibers" can be seen on histology 4-12 hrs post-MI (non-contractile muscle fibers being pulled by adjacent contractile fibers)
- 24. **Blood spots**: punctate hemorrhages can be seen on histology 4-12 hrs post-MI
- Catacomb necropolis: early signs of coagulation necrosis can be seen on histology 4-12 hrs post-MI
- 26. Full-sun mask: histopathology of MI 12-24 hrs (~1 day)
- 27. Full-sun jester falling into catacombs: frank coagulation necrosis can be seen on histology 12-24 hrs post-MI (pale cardiomyocytes, loss of nuclei, preserved structural outline)
- 28. Contracting arms and band pattern on tunic : contraction bands can be seen on histology 12-24 hrs post-MI (return of blood flow →abnormally high intracellular Ca2+ →hypercontraction of dead cardiomyocytes)
- 29. First responders rushing in : neutrophils can be seen in the area of infarct on histology 12-24 hrs post-MI
- 30. 3 Sun buttons: histopathology of MI 1-3 days
- Jester deep in catacombs: extensive coagulation necrosis can be seen on histology 1-3 days post-MI
- 32. **First responders rushing in**: neutrophils become more abundant in area of infarct on histology 1-3 days post MI
- Blue dots on pale tunic : neutrophilic infiltrate between pale cells on histology 1-3 days post MI
- 34. **Red lute case over catacombs**: early onset pericarditis can occur 1-3 days post-MI (ONLY over the area of infarct)
- 35. Shark tooth necklace : early onset (peri-infarct) pericarditis presents with pleuritic chest pain (worse on inspiration and better with leaning forward)
- 36. **Friction markings**: early onset (peri-infarct) pericarditis presents with a pericardial friction rub on physical exam

Cardiac Pathology: Chapter 1.3 Continued



- 37. Half-moon shield: histopathology of MI 3-14 days (~1/2 month)
- Cage transporter: between days 3-14, macrophages infiltrate the area of necrosis
- Tunic rife with blue dots: numerous macrophages infiltrating on histology 3-14 days post MI
- 40. **Granny with tissue**: granulation tissue can be seen on histology 3-14 days post MI (activated myofibroblasts and vascularity)
- Granny in vascular dress: granulation tissue is accompanied by neovascularization 3-14 days post MI
- 42. **Ruptured string holder**: ischemia of the papillary muscle causes necrosis and rupture 3-14 days post-MI
- 43. Regurging jester with bicuspid hat : rupture of papillary muscle and chordae tendinae → mitral regurgitation 3-14 days post-MI
- 44. **Murmur lines from bottle spray**: mitral regurgitation presents with a holosystolic blowing murmur that radiates to the left axilla
- 45. **Jester with wet suit**: mitral regurgitation causes pulmonary edema and worsening dyspnea 3-14 days post-MI
- 46. **Posterior descending jester hat**: papillary muscle rupture can occur with occlusion of the posterior descending artery (branch of right coronary) which perfuses the posteromedial papillary muscle
- 47. Left anterior descending jester hat: interventricular septum rupture can occur with occlusion of the LAD (branch of left coronary) which perfuses the anterior 2/3
- 48. Ruptured tambourine: ischemia of the interventricular septum causes necrosis and rupture 3-14 days post-MI
- 49. **Jester spitting spray**: interventricular septum rupture presents with a holosystolic murmur
- 50. **Ruptured lute body**: ischemia of the ventricle causes necrosis and rupture 3-14 days post-MI
- 51. Left side of jester hat: ventricular free wall rupture can occur with occlusion of the left coronary (or its branches) which perfuses the anterior ventricular wall and apex

- 52. **Guitar case full of water**: ventricular free wall rupture leads to massive hemopericardium and tamponade
- 53. **Lightning heart sigil on cage**: any form post-infarct muscle rupture (3-14 days post-MI) can cause hypotension and cardiogenic shock
- 54. **Cork bouncing off big scar knight** : after 14 days post-MI, fibroblasts (activated by macrophages) deposit collagen and fibrotic tissue → scar formation → decreased risk for rupture
- 55. Quivering heart held by scar knight: scar formation (after 14 days post-MI) can cause conduction abnormalities and fatal ventricular arrhythmias (sudden cardiac death)
- 56. Failing heart balloon: myocardial scar formation can lead to heart failure weeks to months post-MI (decreased contractile function)
- 57. Many moon lanterns: histopathology weeks to months post-MI
- 58. Crossdresser with red lute case : autoimmune (late-onset) pericarditis (Dressler's syndrome) can occur weeks to months post-MI
- 59. Shark tooth necklace: Dressler's syndrome presents with pleuritic chest pain (worse on inspiration and better with leaning forward)
- 60. **Friction mark**ings: Dressler's syndrome presents with a pericardial friction rub on physical exam
- 61. **Flaming dress**: Dressler's syndrome presents with a fever and leukocytosis weeks to months post-MI
- 62. **Antibody arrows shooting at crossdresser**: Dressler's syndrome is an autoimmune pericarditis that results from the formation of IgG autoantibodies against myocardial antigens exposed during acute MI
- 63. **Peasant caving in t**ent: a ventricular wall aneurysm can form weeks to months post-MI due to thinning of a transmural scar
- 64. Failing heart balloon: a ventricular wall aneurysm can cause systolic heart failure
- 65. **Bird nests on wall with embolizing droppings**: ventricular wall aneurysm can cause blood stasis and mural thrombus formation → embolization→ ischemic stroke weeks to months post-MI



- 1. Failing heart balloon: congestive heart failure
- 2. Systolic spray: systolic HF (ejection dysfunction)
- 3. Falling cake fraction: HF with reduced ejection fraction (systolic HF)
- 4. **40 shaped cake server:** ejection fraction <40% (reduced ejection fraction)
- 5. Ultrasound horn: use cardiac ultrasound to measure ejection fraction
- Weak arm lifting heart: reduced cardiac contractility → reduced ejection fraction (systolic HF)
- 7. **High pressure balloon :** ↑end diastolic PRESSURE (seen in systolic HF)
- 8. **High volume balloon:** ↑ end diastolic VOLUME (seen in systolic HF)
- 9. Diamond tiara: diastolic HF
- 10. Difficulty filling glasses: diastolic HF (filling dysfunction)
- 11. **55 cake topper:** ejection fraction >55%(preserved ejection fraction)
- 12. Lifting cake fraction: HF with preserved ejection fraction (diastolic HF)
- 13. Fallen compliance papers: reduced ventricular compliance seen in diastolic HF
- 14. **High pressure balloon:** ↑ end diastolic PRESSURE with normal diastolic volumes(seen in diastolic HF)
 - Dysfunctions that cause systHF
- 15. **Clogged coronary crown:** coronary artery disease (CAD) can cause cardiac ischemia →↓ contractility → systolic HF
- 16. Broken heart string: ACS (e.g. Acute myocardial infarction) can cause myocardial scarring → ↓ contractility→ systolic HF
- 17. **Dilated heart sac:** dilated CMP causes ↓ contractility→ systolic HF
- 18. Regurgitating drink: valvular insufficiency (e.g. Aortic or mitral regurg) can cause volume overload → systolic HF
- 19. **Unbuttoned shirt:** LtoR shunt (e.g. VSD) can cause volume overload \rightarrow systolic HF
- Dysfunctions that cause diasHF
- 20. Stenotic aortic hat (she pulls down her hat=stenosis): AS can cause pressure overload →ventricular hypertrophy→ diastHF
- 21. **High pressure steam:** long standing HTN can cause pressure overload →LV hypertrophy→diastolicHF(most common cause)

- 22. **Big obstructed bagpipes:** hypertrophic obstructive CMP causes ventricular hypertrophy →diasHF
- 23. **Heart in restricted net:** restricted CMP causes a non-compliant ventricle→diasHF
- 24. **Clogged coronary crown:** coronary artery disease (CAD) can cause cardiac ischemia→ noncompliant ventricle→diasHF
- Constricting bow(heart like gift): constrictive pericarditis causes a non-compliant ventricle→diasHF
- Histology systHF
- 26. Pulling load(pulling by a jacket): systolic HF is associated with↑PRELOAD(volume-overload states)
- Long eccentric eel: eccentric hypertrophy (sarcomeres add in series) in response to volume-overload states→systolicHF
- 28. **Dilated cave:** systolic HF is associated with ↑ chamber size (due to eccentric hypertrophy)
 - Histology diasHF
- 29. Pushing load: chronic HTN and valvular stenosis cause diastolic HF by ↑ afterload
- 30. **Concentric conch shell:** concentric hypertrophy (sarcomeres add in parallel) in response to ↑ afterload (e.g. HTN stenotic valve) →diastHF
- 31. **Small shell opening:** diastHF with concentric hypertrophy is associated with ↓chamber size and ↑wall thickness
- 32. **Bulging septum(heart tube):** HOCM causes distHF with isolated septal hypertrophy
- 33. **Normal cardiac bow:** restricted CMP and constrictive pericarditis cause diastHF with normal chamber size and wall thickness



- 34. **Dilated BNP blimp:** brain natriuretic peptide(BNP) is released by stretched CMP in the ventricles
- 35. ANP flag: atrial natriuretic peptide(ANP) is released by stretched CMP in the atria
- 36. **Dilated sleeves:** ANP and BNP cause vasodilation \rightarrow \downarrow afterload(\downarrow SVR)
- 37. **Salty Na peanuts in water:** ANP and BNP promote natriuresis→diuresis→↓preload
- 38. Falling rain umbrella: ANP and BNP ↓renin production—↓aldosterone—natriuresis/diuresis
- 39. **Pinched efferent straw:** ANP and BNP causes efferent arteriolar vasoconstriction→increase GFR→natriuresis/diuresis
- Physiological changes in response to HF
- 40. Increased ground filtration rate (waiter counting his tips): ANP and BNP = ↑GFR
- 41. "OUTPUT LOW" HF is associated with \downarrow CO \rightarrow compensatory mechanism
- 42. **Fight or flight activator**: ↓CO causes ↑sympathetic activation →↑HR and cardiac contractility
- 43. **Twisted arterial sleeve:** ↓CO causes ↑sympathetic activation →vasoconstriction(↑SVR)
- 44. Rain umbrella and tight red suspenders: ↓CO causes ↑renin activity →↑ angiotensin II→ vasoconstriction (↑SVR)
- 45. Pulsing load (guy with umbrella pushing on the door) sympathetic activation ↑afterload (vasoconstriction)
- 46. **Wet rain umbrella and salty minerals:** ↓CO causes ↑renin activity → ↑aldosterone → salt and water retention
- 47. Water refill: ↓ CO causes ↑ ADH activity → ↑free water retention
- 48. Wet lifer preserved and peripheral pants: compensatory mechanism of HF (e.g. RAAS activation, ADH activation) exacerbate pulmonary and peripheral edema
- 49. "REMODELING": long-term neurohormonal activation (RAAS, ADH, sympathetic) + ↑hemodynamic stress (HR/contractility, vasoconstriction, extracellular volume) →deleterious cardiac remodeling



Left side HF -left side of sketch-

- 1. Wet life vest prince: left-side heart failure
- 2. Wet pulmonary vest: pulmonary edema: left-sided HF)
- Pink sea foam in abalone shells: frothy pink transudate on the intraalveolar surface (left-side HF)
- Rusty macro-cages: hemosiderin-laden alveolar macrophages HF cells (L side HF)
- 5. Restrictive corset: pulmonary edema reduces pulmonary compliance
- 6. Difficult breath: HF can cause dyspnea with exertion
- 8. Reclining into water: orthopnea (left sided HF)
- Gasping awake(guy in canal): paroxysmal nocturnal dyspnea (PND-left sided)
- Bilateral slurping snorkels: bibasilar inspiratory crackles(left-sided HF)(sound like slurping soda)
- 11. Wheezy party blower: peribronchial edema causes wheezing (left sided HF)(known as like "cardiac asthma")
- 12. Skull and X bones: chest xray(imaging for suspected left sided HF)
- 13. White branches over the top sails: cephalization of the pulmonary vessels on CXR (left sided HF)
- 14. infiltrating fog: pulmonary edema looks like fluffy bilateral ("batwing" shape) opacities on CXR)(left sided HF)
- 15. Curly letter B: Kerley B lines (fluid accumulation between lobes) on CXR(left sided HF)
- 16. Shadow of captain on sail: air bronchogram(dark airway against opacified interstitium) on CXR (left sided HF)

- 17. Big heart: cardiomegaly on CXR (HF)
- 18. **"slushi3":** s3 hear sound(more common in systolic HF) (comes <u>after</u> s1 and s2; early diastole) sound like "slushing in", "slushing in", slushing in", slushing in", slushing in", slushing in"
- 19. Stiff s4 chair: S4 heart sound (more common in diastolicHF)(comes before s1 and s2; late diastolic); sound like "a stiff wall", "a stiff wall"
- Systolic spray murmur: left sided HF can present with a systolic murmur (mitral regurg)
- 21. **Regurgitating mitral hat jester:** dilation of mitral annulus→ mitral regurg(left sided HF)
- 22. **Dilated balloon:** dilated atrium(due to left-sided HF(when mitral valve is open up and blood going in wrong direction→chronic dilation of LA)
- 23. Irregularly irregular signal: Afib(due to atrial dilation in HF)
- 24. Left side HF can damage to endothelium lining of pulmonary vasculature
- 25. **Damaged NO exhaust:** left-sided HF causes dmg to the pulmonary vascular endothelium $\rightarrow \downarrow$ NO \rightarrow vasoconstriction
- 26. **Twisted arterial shirt:** dmg to the pulmonary vascular endothelium → ↓NA and ↑endothelins →vasoconstriction
- 27. **Smooth muscular shark tattoo:** pulmonary vascular remodeling→collagen deposition (intimal hypertrophy) and smooth muscle cell proliferation(medial hypertrophy)
- 28. **Tense pulmonary tree:** left-sided HF →pulmonary artery HTN→Right sided failure(most common cause)



Right side HF

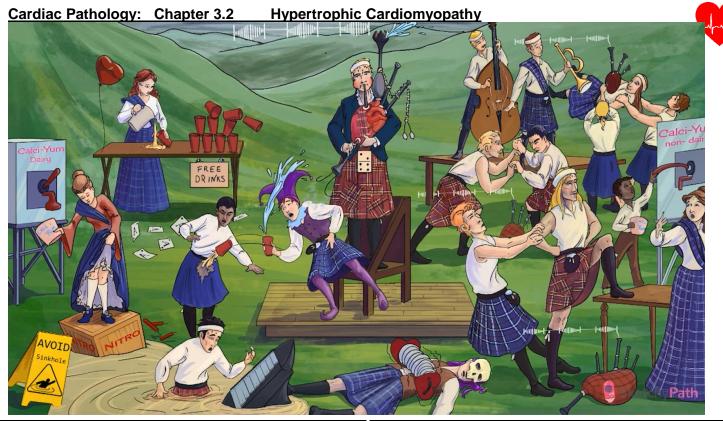
- 29. Cork on the bottle: cor pulmonale (right-sided HF due to pulmonary HTN)
- Embolic sea-cucumbers: chronic pulmonary emboli→cor pulmonale(right-sided HF)
- 31. **Embolic saddle:** a saddle pulmonary embolism→ right heart strain and failure
- 32. Little mermaid pushing her father=pushing load: RV works against an ↑ afterload (cor pulmonale)
- 33. When RV fails →pressure ↑ and stretch open fibrous ring attached to tricuspid valve→tricuspid regurgitation
- 34. **Regurgitating on three peaks:** dilation of tricuspid annulus→tricuspid regurg (right-sided HF)
- 35. **Systolic spray murmur:** right-sided HF can present with systolic murmur (tricuspid regurg)
- 36. More pressure in RA→backup to venous system
- 37. **Distended blue jug:** jugular vein distention (JVD) seen in right-sided HF
- Liver knocking over distended jugular hepato-jugular reflux(rightsided HF)(pressure to RUQ)
- 39. <u>Kaussmal sign</u>(when you breath in lung sucks in blood into pulmonary vasculature, all this blood is coming from right side of heart, so the JV empty as well. This is normally seen as ↓Jugular vein distention during inspiration

- 40. Cookie smell from distended jug: Kussmaul sign (JVD ↑ during inspiration) seen in right-sided HF
- 41. **Swollen sweat pants(mermaid's):** peripheral edema of lower extremities (right-sided HF)
- 42. Wet pleural shirt: pleural effusion (right-sided HF)
- 43. Wet heart case: pericardial effusion (right-sided HF)
- 44. **Course III:** zone3 (centrilobular) necrosis due to hepatic venous congestion (right-sided HF)
- 45. **Nutmeg sprinkled liver:** centrilobular necrosis apprears as "nutmeg liver" on gross pathology
- 46. Painful liver spot: hepatic congestion causes painful hepatomegaly (right-sided HF)
- 47. High pressure porthole: portal HTN seen in right-sided HF If portal pressure ↑→fluid leaks out into peritoneal cavity
- 48. swollen inner tube: ascites due to portal HTN (right-sided HF)



- 1. Dilated heart sack: dilated Cardiomyopathy (CMP)
- 2. Idiot sack racer: dilated CMP is most commonly idiopathic
- 3. Dilated heart sack on fire: myocarditis (e.g. Due to viral infection) can cause dilated CMP
- 4. Systolic spray: dilated CMP causes SYSTOLIC HF
- Failing heart balloon: dilated CMP causes contractile dysfunction →SystolicHF
- Viral lantern igniting sack: viral myocarditis (e.g. Due to coxsackievirus type B) can cause dilated CMP
- Preceding viral lanterns: viral myocarditis can be preceded by flu-like symptoms (e.g. Fever, runny nose, myalgia)
- 8. "sCottisH GAmeS": Chagas dis can cause dilated CMP
- Genetic pedigree tartan pattern: genetic mutation are 2nd most common cause of dilated CMP
- 10. Domino clap: hereditary dilated CMP is usually caused by an autosomal dominant mutation
- 11. Cytoskeleton sack pattern: genetic causes of dilated CMP include mutation in cytoskeletal protein
- 12. Duchenne and Becker clans: Duchenne and Becker muscular dystrophy (Xlinked) can cause dilated CMP
- "Destroy" Duchenne and Becker muscular dystrophy are caused by a mutation in the protein dystrophin (connects myocyte cytoskeleton to the extracellular matrix)
- 14. "box-o-rubies" doxorubicin (anthracycline chemotherapeutic) can cause free radical dmg and cardiotoxicity →dilated CMP
- Alcoholic sack racer: alcohol abuse can cause cardiotoxicity(due to EtOH and acetaldehyde)—dilated CMP
- 16. Bunch of blueberries: alcohol abuse can cause wet beriberi(alcohol to thiamine def) —dilated CMP
- 17. Pregnant sack racer: pregnancy can cause dilated CMP(peripartum CMP)
- 18. Sack with Iron weight: hemochromatosis can cause iron deposition in cardiomyocytes—dilated CMP(or restrictive)
- 19. "slushi3" slushy: dilated CMP can present with an s3 heart sound
- 20. **wall nests**: mural thrombi may be present in dilated CMP→thromboembolism and embolic stroke

- 21. **Bull horn sound**: USG can be used to dx dilated CMP (enlarged heart with dilation of all 4 chamber)
- 22. Heart in restrictive net: restrictive CMP
- 23. Falling compliance rulebook: restrictive CMP is associated with \downarrow ventricular compliance
- 24. Diamond tiara: restrictive CMP causes DIASTOLIC HF(
- 25. difficulty filling glasses: diastolic HF (filling dysfunction)
- 26. Stiff S4 chair: restrictive CMP can present with an S4 heart sound
- 27. Distended blue jug: JVD seen in restrictive CMP
- 28. "Falling Y" glass: restrictive CMP can present with a prominent Y descent (in the JVP waveform)
- 29. Cookie smell from distended jug: Kussmaul sign (JVD ↑ during inspiration) seen in restrictive CMP
- 30. **Armored lady in light chainmail**: amyloidosis (e.g. Light chain or transthyretin deposition) can cause restrictive CMP
- 31. **Amorphous pink areas**: endomyocardial biopsy shows areas of amorphous and acellular pink material in the myocardium (H&E stain)
- Bright green skirt: cardiac amyloidosis shows apple-green birefringence under polarized light microscopy (congo red stain)
- 33. Soccer balls: sarcoidosis can cause restrictive CMP(present with noncaseating granulomas presenting with multinucleated giant cells)
- 34. **Iron weight toss**: hemochromatosis can cause Fe deposition in cardiomyocytes → restrictive CMP(usually dilated) (blue face=prussian blue detection)
- 35. **Spillin garbage truck**: lysosomal storage dis (e.g. Hurler's syndrome, Fabry's dis) can cause restrictive CMP
- 36. Scaly red dragon: scleroderma can cause restrictive CMP(systemic sclerosis)
- 37. Radiation shield: chest irradiation can cause fibrosis of the myocardium→restricitve CMP
- 38. Fibrous heart log: endomyocardial fibrosis (prevalent in tropical regions) can cause restrictive CMP
- 39. Fibrous heart log with elastic band holding lose kilt in a young boy: endomyocardial fibroelastosis (in infants) can cause restrictive CMP



- 1. Big obstructed heart bag: hypertrophic cardiomyopathy (HOCM)
- 2. **Wide septal stripe on heart bag**: the interventricular septum shows the most significant amount of myocardial hypertrophy in HOCM
- 3. **Obstructive knot**: the massive IVS in HOCM can obstruct blood flow out of the left ventricle
- 4. **Difficulty filling glasses**: massive septal hypertrophy in HOCM causes diastolic dysfunction (small chamber size)
- 5. Failing heart balloon: HOCM can cause diastolic heart failure
- 6. **Domino sporran**: HOCM is caused by an autosomal dominant mutation of sarcomere proteins
- 7. **b-myosin rope on pipes**: HOCM is commonly caused by a *gain of function* mutation in sarcomere proteins (e.g. b-myosin heavy chain, myosin binding protein C, troponin T) \rightarrow increased myofilament activity and hypertrophy
- 8. **Disorganized plaid pattern**: the gain of function mutations in sarcomere proteins cause disorganized myofibrillar proliferations
- Dead musician with quivering heart: aberrant myofibers cause aberrant conduction pathways → fatal arrhythmias (e.g. VT, VF) and SCD
- 10. Athletic sweatband: HOCM usually manifests before puberty and is most common cause of sudden death in young athletes
- 11. **Obstructive knot below valve**: the obstruction to blood flow from the LV in HOCM occurs below the aortic valve in the LVOT
- 12. **Bicuspid jester hat blown forward**: systolic anterior motion of the mitral valve (and its contact with the hypertrophied interventricular septum) causes LVOT obstruction in HOCM
- 13. **Spilling on bicuspid hat**: contact of the anterior leaflet of the mitral valve and the interventricular septum causes the mitral valve to remain open during systole \rightarrow mitral regurgitation
- 14. **Murmur from obstructed pipe**: HOCM can present with a harsh, crescendo-decrescendo systolic murmur best heard at the left sternal border (caused by LVOT obstruction)
- 15. Standing and straining bagpiper: valsalva and standing DECREASE preload \rightarrow higher degree of LVOT obstruction \rightarrow INCREASED murmur intensity
- 16. **Squatting leg raise**: leg raise (when supine) and squatting INCREASE preload and ventricular size \rightarrow smaller degree of LVOT obstruction \rightarrow DECREASED murmur intensity
- 17. **Pulling load**: maneuvers that INCREASE preload (e.g. squatting, leg raise) DECREASE murmur intensity
- 18. **Grappling and squatting**: squatting and handgrip maneuvers increase SBP and afterload \rightarrow slow movement of blood through LVOT \rightarrow DECREASED murmur intensity

- 19. **Pushing load**: maneuvers that increase afterload (e.g. squatting, handgrip) DECREASE the murmur intensity
- 20. **Stiff S4 chair**: HOCM can present with an S4 heart sound (blood hitting the stiff noncompliant ventricle)
- 21. **Sinking in quicksand (sink hole)**: HOCM can cause syncope from non-lethal arrhythmia or temporarily decreased CO
- 22. **Angina anvil**: HOCM can cause angina (hypertrophic tissue impedes subendocardial blood flow)
- 23. **Muted bugle**: HOCM can be treated with *b-blockers* (decrease inotropy and chronotropy)
- 24. **Floppy bass strings**: b-blockers treat HOCM by decreasing cardiac contractility (decreased inotropy) → slow blood flow across LVOT obstruction -also, it just decreases myocardial oxygen demand
- 25. **Non-dairy Calci-Yum ice cream**: non-dihydropyridine calcium channel blockers treat HOCM by decreasing cardiac contractility (decreased inotropy) → slow blood flow across LVOT obstruction
- 26. **Pulling load**: b-blockers and non-dihydropyridines Ca Blockers treat HOCM by decreasing HR \rightarrow increased time in diastole and left ventricular PRELOAD \rightarrow decreased LVOT obstruction
- 27. **Avoid sinkhole**: several medications are C/I in HOCM (e.g. drugs that decrease preload such as diuretics, nitrates, or dihydropyridines; and drugs that increase contractility such as digitalis or milrinone) \rightarrow increase obstruction
- 28. **Dairy Calci-Yum ice cream**: avoid dihydropyridine in HOCM (vasodilation decreases afterload → increased velocity of blood in LVOT → increased obstruction) → increased pressure gradient
- 29. **Dilated arterial sleeves**: avoid vasodilators in HOCM (vasodilation decreases afterload \rightarrow increased velocity of blood in LVOT \rightarrow increased obstruction) \rightarrow increased pressure gradient
- 30. **Sinking nitro box**: avoid nitrates in HOCM (venodilation decreases preload and left ventricular size → increased LVOT obstruction)
- 31. **Falling aces**: avoid ACE inhibitors in HOCM decreases preload and afterload (AGII) \rightarrow increased LVOT obstruction) \rightarrow less aldosterone also decreases preload
- 32. **Wet crotch**: avoid diuretics in HOCM (decreases preload and left ventricular size → increased LVOT obstruction)
- 33. **Toppling free drinks**: Friedreich' (AR trinucleotide repeat disorder that causes ataxia and cardiomyopathy) is associated with HOCM (the most common cause of death from Friedreich's ataxia)



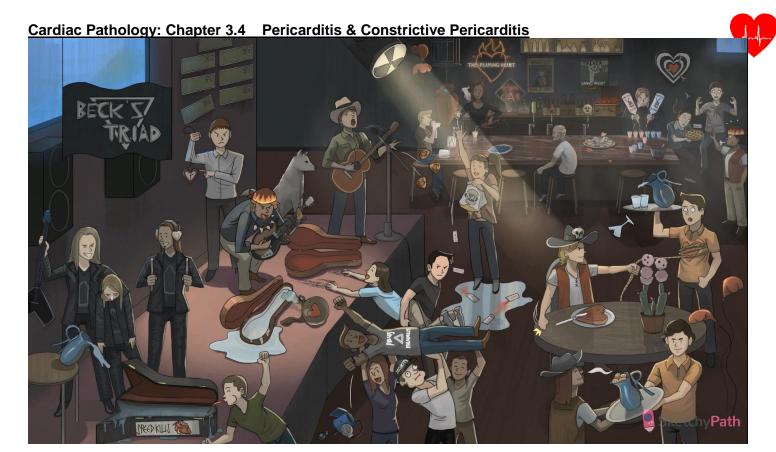
- The Flaming Heart: myocarditis (inflammatory dmg to myocardium caused by infection, toxin exposure, or hypersensitivity reaction)
- Cockatoo: infection with Coxsackie B (enterovirus can cause myocarditis)
- Scattered blue bird seed: viral myocarditis show an inflammatory infiltrate in the myocardium on histology
- 4. Spilled Ab toothpicks: viral damage to myocytes causes the release of cross-reactive Ag→ Ab target heart tissue (viral myocarditis involve direct viral injury to myocytes as well as further inflammation caused by own Ab response)
- 5. Dilated heart sack: myocarditis can lead to dilated CMP
- 6. Failing heart balloon: myocarditis can lead to systolic HF
- 7. **Blowing nose**: viral myocarditis may be preceded by flu-like symptoms(~1 week prior)
- 8. **The Flaming Heart**: myocarditis (inflammatory dmg to myocardium caused by infection, toxin exposure, or hypersensitivity reaction)
 - +Nonviral causes
- Che's gAs: Chagas disease (infection with the protozoan Trypanosoma cruzi) may include myocardial involvement

 myocarditis and dilated CMP
- Protozoal bar nuts: T.cruzi infection of myocardium shows dense collection of protozoa on histology
- 11. **Bacterial beer tap handle**: bacterial infection (e.g. Borrelia, Rickettsia, Mycoplasma) can cause myocarditis
- 12. "Robin of Ixodes": Lyme diseases (infection with the bacterium Borrelia burgdorferi) can include myocarditis
- 13. **Heart shield**: myocardial involvement in Lyme disease can manifest as heart block
- 14. Fungus beer tap handle: fungal infection(e.g. Candida, Mucor, Aspergillus) can cause myocarditis
- 15. **Immunocompromised cane**: fungal myocarditis is more common in immunocompromised
- 16. Toxin beer tap handle: toxins exposure (alcohol, carbon monoxide, cocaine, diuretics, abx) can cause myocarditis
- Chips and dip: Corynebacterium.diphtheriae toxin can cause myocarditis
- 18. **Box of rubies**: anthracyclines (e.g. Doxorubicin/daunorubicin) can cause free radical damage and myocarditis

Autoimmunity

- Helper with squires(bottle): certain drugs can elicit a delay type IV hypersensitivity reaction (helper T cell mediated) → hypersensitivity myocarditis
- 20. **Mortar and pestles**: drugs that cause hypersensitivity myocarditis include sufla drugs, furosemide, HCTZ, ampicillin, azithromycin and zidovudine
- Blue & pink shot glasses: hypersensitivity myocarditis manifests histological with lymphocytic and eosinophilic interstitial inflammatory infiltrates
- 22. **AB darts**: autoimmune disease (e.g. SLE, scleroderma, and RA) can cause myocarditis
- 23. **Rhubarb pie**: Acute Rheumatic Fever is associated with myocarditis (~2-4 weeks after Strep pyogenes pharyngitis via molecular mimicry)

 Clinical Signs
- 24. Hot dyspneic dark thrower: myocarditis often presents with constitutional symptoms such as fever, malaise, and dyspnea
- 25. **Dark in chest**: myocarditis can produce a precordial chest pain that can mimic MI
- 26. Bucket of Chicken and T Bone: inflammation and myocardial damage from myocarditis can cause release of troponins and CK MB
- 27. Passed out with vibrating heart: abnormal cardiac conduction from inflammation in myocarditis can lead to fatal arrhythmia and sudden cardiac death



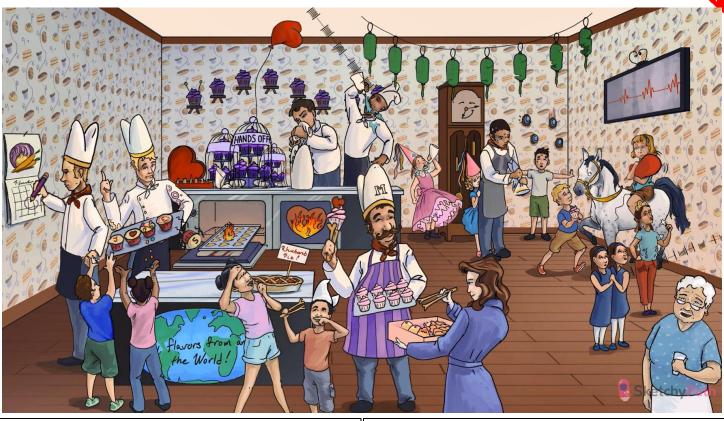
- 1. Red heart case: pericarditis
- Flaming heart bar: many of the causes of myocarditis (e.g. viral infection, bacterial infection, toxins, and autoimmune disorders) can also cause pericarditis
- 3. **Wolf on stage**: pericarditis is one of the most common clinical features of systemic lupus erythematosus
- Broken heart string: MI causes two forms of pericarditis, an initial inflammatory form and an autoimmune form that follows 2-4 weeks later (Dressler syndrome)
- 5. Elevated BUN bag: uremia can cause pericarditis
- Throwing crab picks: metastases to the pericardium can cause pericarditis
- 7. **Guitar player leaning forward with flame bandana**: fever is a common constitutional sign seen in pericarditis
- 8. Elevated heart watch: pericarditis can present with tachycardia
- Leaning forward with shark tooth necklace: the pain of pericarditis is characteristically precordial and sharp; worsened with inspiration and relieved with leaning forward
- Friction marks: movement of the inflamed visceral and parietal pericardial layers against each other creates a friction rub on auscultation
- 11. **Bunch of elevated street signs**: pericarditis causes a characteristic diffuse ST elevation on ECG
- 12. Wet heart case: pericarditis can cause pericardial effusion
- 13. **Ketchup packets in puddle**: conditions such as uremia or malignancy can cause hemorrhagic pericarditis
- 14. Guitar case filled to brim: if a pericardial effusion becomes severe enough, it can impair cardiac filling and preload
- 15. "Speed kills": the rate of pericardial filling (not necessarily the volume) that determines severity of effusion (and morbidity in cardiac tamponade)
- 16. Fist squeezing heart : cardiac tamponade (rapidly developing pericardial effusion that limits the heart's ability to expand and fill in diastole)
- "Beck's triad": cardiac tamponade presents with muffled heart tones, hypotension, and JVD (Beck's triad)
- Distended blue jug: tamponade can cause jugular vein distension (JVD) (compression of the heart raises right atrial pressure, impairing filling)

- 19. **Passed out**: cardiac tamponade can present with hypotension (and in extreme cases cardiogenic shock or death)
- 20. **Heart earmuffs**: cardiac tamponade presents with muffled heart sounds
- 21. "Pulsus paradoxus" : cardiac tamponade can present with pulsus paradoxus (>10mm drop in SBP on inspiration)
- --Equally distributed weight: pulsus paradoxus is caused by all heart chambers having equal pressure due to extrinsic compression --Bowing to the left with inspiration: increased venous return during inspiration with a non-distensible right ventricle causes it to bow into the
- 22. **BP cuff falling**: in cardiac tamponade, inspiration can cause a drop in systolic blood pressure >10 mmHg (pulsus paradoxus)

left ventricle → decreased LV chamber size and stroke volume

- 23. **Straw in guitar case**: the only treatment for severe cardiac tamponade is drainage via pericardiocentesis
- 24. Lasso around heart : constrictive pericarditis (scarring and fibrosis from pericarditis can leave the pericardium stiff and non-distensible → limited expansion during diastole)
- 25. **Distended blue jug**: constrictive pericarditis cause JVD (due to impaired right atrial filling)
- 26. Y **shaped falling glass**: constrictive pericarditis causes a prominent Y descent on the jugular venous waveform (due to rapid atrial emptying)
- 27. Sniffing cookie smell from jug: inspiration causes an increase in JVD in constrictive pericarditis (Kussmaul's sign)
- 28. Failing heart balloons: constrictive pericarditis can cause diastolic heart failure and symptoms of CHF
- 29. **Knocking on table**: ventricles expanding against a thickened pericardium in constrictive pericarditis causes a characteristic "pericardial knock" on auscultation
- 30. White brim: in chronic constrictive pericarditis, calcification of the pericardium is seen as a white rim around the heart on CXR or CT
- 31. Cavitary TB cactus: constrictive pericarditis can be caused by M. tuberculosis infection (tuberculous pericarditis)
- 32. **Knife in heart**: a common cause of constrictive pericarditis is scarring following open heart surgery
- 33. **Radiation symbol**: thoracic radiation for conditions such as breast or lung cancer can lead to constrictive pericarditis

Cardiac Pathology: Chapter 4.1 Acute Rheumatic Fever Mitral Stenosis (Rheumatic Heart Disease)



- 1. Bicuspid chef hat: ARF primarily affects mitral valve
- Rhubarb pie: rheumatic fever (ARF- acute multisystem inflammatory disease that can follow a group A Strep infection)
- 3. Striped pie chef: Strep pyogenes (group A Strep)
- Scrumptious Stenosis Acute Rheumatic Fever Mitral Stenosis (Rheumatic Heart Disease)
- Red neck kerchief: streptococcal pharyngitis can lead to rheumatic fever (NOT skin or other GAS infection)
- 6. World map: ARF is prevalent in underdeveloped countries
- 7. Kids: ARF most often affects children between 5-15 years old
- Later in the month(chef marking calendar): ARF usually develop ~2-3 weeks after strep pharyngitis
- Antibody tongs: cardiac damage in ARF is caused by a type II hypersensitivity reaction (Ab mediated)
- 10. **Kid mimicking chef**: Ab in ARF are formed by molecular mimicry
- JONES cupcakes: JONES criteria for dx ARF Joints (Migratory polyarthritis) O (myocarditis) Nodules (sub-q) Erythema marginatum, Sydenham chorea
- 12. "J" with frosting on elbows: ARF commonly presents with migratory polyarthritis (usually large joints such as the elbow, knees, and ankles)
- "O" heart: ARF can cause pancarditis affecting pericardium, myocardium and endocardium(valves)
- 14. "N" with nodular candies: ARF can present with subcutaneous nodules (form mostly on extensor surface of forearm & may show central fibrinoid necrosis)
- 15. "E": AFR present with a rash that consists of hove like C-shaped area of erythema
- 16. "S" falling: "Sydenham chorea" → ARF present with rapid involuntary movements affecting all muscle throughout the body (may show up 1-8 months after infection)
- Pan of heart cookies: ARF can cause pancarditis affecting pericardium, myocardium and endocardium(valves)
- 18. Red heart case: ARF can cause pericarditis
- Heart on fire: ARF can cause myocarditis (most common cause of death)
- Failing heart balloon: ARF induced myocarditis can cause acute heart failure (pulmonary and peripheral edema in a young person)

- 21. Multi-cupcake cage: granulomas composed of macrophage, multinucleated giant cells, lymphocytes and plasma cells can be found in any layer of heart in ARF
- 22. "hand off" Aschoff bodies (characteristic granulomas histological finding in ARF)
- Caterpillar cupcakes: Anitschkow bodies ("caterpillar") cells(activated macrophages with slender, ribbon like nuclei) maybe be seen in granulomas of ARF
- 24. Flame in heart: ARF can cause endocarditis -specifically a valvulitis-
- Frosting on bicuspid hat: ARF can cause fibrinoid necrosis and sterile verrucous vegetation on the line of valve leaflet closure (mitral most common)
- 26. Regurgitation bicuspid hat chef: valve damage in ARF can cause Mitral regurgitation
- 27. Murmur lines from regur: ARF can present with a new-onset harsh holosystolic murmur over the apex that radiated to left axilla (mitral regur)
- 28. Regurgitation aortic princess hat: valve damage in ARF can cause aortic regurgitation(mitral more common)(diastolic= diamond)
- 29. Lysed jelly donuts and eaten helix donut: Antistreptolysin-O and anti-DNase B titers can be used to dx previous strep infection in ARF (cultures may be negative by the time pts present)
- 30. **Purple pencil**: penicillin tx ARF (sometimes given for years depending on severity of carditis)

Cardiac Pathology: Chapter 4.1 Acute Rheumatic Fever Mitral Stenosis (Rheumatic Heart Disease)



- Recurring bacterial lanterns: subsequently GAS infection cause repeat episodes of ARF and worsening symptoms →chronic rheumatic heart dis
- 32. Chronic grandfather clock: pts may present years later with rheumatic heart disease. Due to chronic damage and repair → chronic rheumatic heart disease
- 33. Chef wringing bicuspid hat: years of inflammation and scarring of the mitral leaflets in chronic RHD can lead to mitral stenosis
- 34. **Stenotic princess (behind chef)** chronic RHD may also present with aortic stenosis (mitral more common)
- 35. **Bulging heart balloon**: mitral (or aortic) stenosis can cause left atrial (LA) dilation (LA has to pump blood through tiny stenotic opening→ pressure↑ →LA dilation)
- 36. Irregularly irregular signal: LA enlargement can lead to atrial fib
- Mural cupcakes: LA enlargement and A fib can cause blood stasis and mural thrombus formation
- 38. Chocolate spots on head: mural thrombi in LA enlargement can embolize→ischemic stroke
- Recurrent reigns on horse: compression of left recurrent laryngeal nerve by a dilated LA can cause chronic cough or hoarseness
- 40. **Gulping(horse):** compression of esophagus by a dilated LA can lead to dysphagia and regurgitation of food

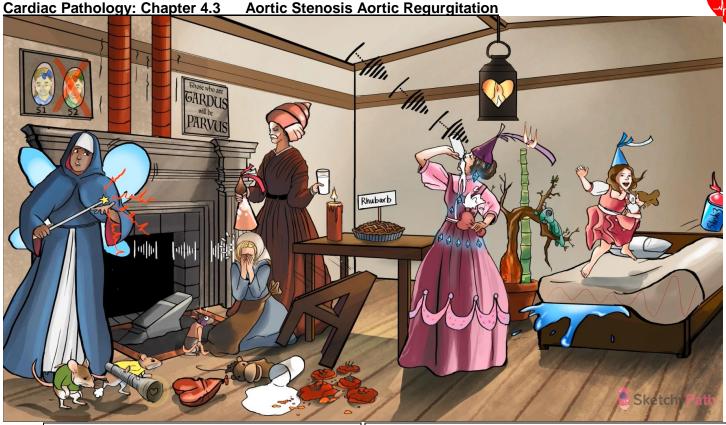
- 41. **Sweaty shirt(stenotic chef)** Mitral Stenosis→ ↑LA pressure →symptoms of left heart failure (e.g pulmonary edema)
- Diamonds and rumbling stomach: Mitral stenosis presents with a mid-diastolic rumbling murmur
- 43. "Snap!" mid-diastolic murmur of Mitral Stenosis is preceded by an opening snap (head over apex of left sternal border)
 --snap is heard during diastole, which means it comes right after s2, when lots of pressure ↑behind stenotic valve it opens sooner in diastole the closer snap is to s2 the more severe stenosis
- 44. **2 scared sisters**: the closer the opening snap is to s2 heart sound, the greater severity of Mitral Stenosis
- 45. Granny with calcifications around the mouth: MS can be also caused by annular calcifications(degenerative calcium deposition in fibrous ring of the mitral valve in older people) though uncommon

Cardiac Pathology: Chapter 4.2 Mitral Valve Regurgitation Mitral Valve Prolapse



- 1. Regurgitating bicuspid jester hat : mitral regurgitation
- 2. Parasailing jester with #1 finger: mitral valve prolapse is the number one cause of mitral regurgitation
- 3. **Mixer on the parasailer**: myxomatous degeneration (pathologic deterioration of connective tissue) causes mitral valve prolapse
- Broken heart string: acute mitral regurgitation can be caused by papillary muscle rupture following MI
- Strings broken on paraglider: myxomatous degeneration can cause chordae tendinae rupture and acute mitral regurgitation
- Flame in heart lantern : infective endocarditis can damage the chordae tendinae → rupture and acute mitral regurgitation
- 7. **Broken heart flashlight** : acute mitral regurgitation decreases forward stroke volume → acute drop in cardiac output
- Pulling load: acute mitral regurgitation increases left ventricular end diastolic volume and PRELOAD
- Wet life vest: acute mitral regurgitation can cause pulmonary venous hypertension and flash pulmonary edema
- Pushing load: acute mitral regurgitation causes a second lowresistance outlet for the ventricle → decreased afterload
- 11. Raised heart fraction: the decreased afterload in acute mitral regurgitation leads to an increased ejection fraction (but lower forward stroke volume)
- 12. Falling lightning bolt batteries: acute mitral valve regurgitation can cause severe hypotension and cardiogenic shock
- 13. Rhubarb pie : acute rheumatic fever causes valvulitis → mitral regurgitation
- 14. Flame in heart lantern : infective endocarditis can cause vegetations to form on the mitral valve → mitral regurgitation
- 15. **Floppy heart bal**loon : heart failure can cause dilation of the left heart chambers and mitral annulus → functional mitral regurgitation (can be reversible)
- Dilated heart bag : dilated cardiomyopathy can stretch the mitral annulus → mitral regurgitation
- 17. Chronic grandfather clock in the dilated balloon: chronic mitral regurgitation allows for the left atrium dilation and hypertrophy→ less pressure transmitted to pulmonary circuit (no significant pulmonary edema)
- 18. **Raised fraction**: an increased ejection fraction maintains cardiac output in chronic compensated mitral regurgitation

- 19. **Pulling load**: chronic mitral regurgitation causes a chronically elevated preload in the left ventricle
- Eccentric myocardial ribbon: chronically increased preload in chronic mitral regurgitation causes eccentric hypertrophy of the left ventricle
- 21. Failing heart balloon: chronic mitral regurgitation can progress to decompensated congestive heart failure
- 22. **Murmur from systolic s**pray: mitral regurgitation presents with a blowing, holosystolic murmur
- 23. **Jester in arm**pit: the systolic murmur of mitral regurgitation is heard best over the cardiac apex with radiation to the axilla
- 24. Hologram: the murmur of mitral regurgitation is holosystolic
- 25. **Sloshing Slus**hi3: dilation of the left ventricle with chronic mitral regurgitation can cause an S3 heart sound
- 26. **Wet crotch closing bicuspid hat**: functional mitral regurgitation (due to acute left ventricular volume overload) can be corrected with diuresis (decreased murmur)
- 27. **Martian with mi**xer: connective tissue diseases such as Marfan syndrome, osteogenesis imperfecta, and Ehlers-Danlos can cause mitral valve prolapse (due to myxomatous degeneration)
- 28. Clicking carabiner between S1 and S2: mitral valve prolapse presents with a mid-systolic click (sudden tensing of the chordae as valve leaflets prolapse) between the S1 and S2 heartsounds
- 29. Straining next to S1: maneuvers that decrease preload (e.g. Valsalva) cause the mid-systolic click in MVP to move closer S1
- Elevated heart watch next to S1: Tachycardia (decreases the diastolic filling time and preload) will cause the mid-systolic click in MVP to move closer to S1
- 31. **Propping legs up next to S**2 : maneuvers that increase preload (e.g. straight leg raise) cause the mid-systolic click in MVP to move closer to S2
- 32. **Squatting next to S2**: maneuvers that increase preload (e.g. squatting) cause the mid-systolic click in MVP to move closer to S2
- 33. Sustained grip next to S2: maneuvers that increase afterload (e.g. sustained hand grip) cause the mid-systolic click in MVP to move closer to S2



- Aortic Stenosis (AS)
- 1. Squeezing aortic princess hat: aortic valve stenosis
- 2. **Milk on lips**: calcific degeneration of aortic valve leaflets is mcc of AS in US
- 3. **Black aortic hat tip:** calcific degeneration of aortic valve is associated with endothelial and fibroblast cell death
- 4. **Clogged arterial candle**: risk factors for calcific degeneration of aortic valve include risk factors for atherosclerosis (eg HTN, hyperlipidemia, DM, inflammation)
- 5. **Crusty tip on candle**: calcific degeneration of aortic valve shows up as fine, gritty white clumps on histology
- 6. **Bicuspid horse mouth next to milk**: bicuspid aortic valves is at risk of early dystrophic calcification
- 7. **Concentric conch**: chronically increased afterload in AS leads to concentric hypertrophy of Left ventricle
- 8. Failing heart balloon: aortic stenosis can cause diastolic HF
- 9. **Dilated left wing**: AS causes chronically elevated pressure in the left ventricle and atrium →LA dilation and hypertrophy
- $10. \ \textbf{Irregularly irregular signals} : AS \ can \ cause \ aFib \ (due \ to \ LA \ dilation)$

Clinical manifestation of AS

- 11. **Mouse huffing and puffing**: AS can present with HF symptoms (dyspnea on exertion)
- 12. **Dim heart light**: AS can cause "fixed CO" (unable to ↑ with stress)
- 13. **Angina anvil**: AS can cause angina (due to a fixed CO and ↑O2 demand from cardiac hypertrophy)
- 14. **Sinking quicksand**: AS can cause syncope (due to fixed CO that cannot ↑ during standing or exercise → ↓brain perfusion)
- $15.\,{\rm Murmur}$ from systolic spray: AS can present with harsh, crescendo-decrescendo holosystolic murmur
- 16. Pair of vibrating pipes: murmur of AS is best heard at right sternal border and radiates to carotid arteries
- 17. **Stiff S4 chair**: concentric hypertrophy in AS lead to LV wall stiffnes→S4 sound
- 18. "Parvus and Tardus": severe AS present with a weak slow-rising pulse ("pulsus parvus et tardus")
- 19. Late bloomer: more sever AS present with a late peak in the crescendo
- 20. **Crossed out second sister**: severe AS present with diminished S2 heart sound (s1=mitral valve closure, s2=aortic closure)

- 21. **Squatting Cinderella**: maneuvers that ↑ preload (straight leg raise, squatting) ↑ murmur of AS (due to ↑ SV across valve)
- 22. **Straining to grip tail**: maneuvers that ↓preload (standing Valsalva) or ↑afterload (handgrip) reduce the murmur of AS (due to reduced SV across the valve)
- 23. **Lysed tomatoes** jet stream through a AS valve can cause hemolytic anemia (schistocytes on blood smear)

Aortic Regurgitation (AR)

- 24. Regurgitating aortic princess hat: AR
- 25. **Rhubarb pie in the center**: valvulitis in ARF can cause AR (mcc in developing world) chronic rheumatic heart dis can cause AS
- 26. **Regurgitating milk**: dystrophic calcification can also cause AR (mcc in developing. World)
- 27. Bulging aortic hat base: aortic root dilation can cause AR
- 28. **Bark on aortic tree**: aortitis in tertiary syphilis ("tree-barking") can cause AR (due to aortic root dilation)
- 29. **Inflamed tree base**: large vessel vasculitides (eg Takayasu arteritis, giant cell arteritis (can cause AR (due to aortic root dilation)
- 30. **Bamboo spine**: Ankylosing Spondylitis can cause AR (due to sclerosis of aortic root)
- 31. **Martian**: collagen vascular dis (eg. Marfan, Ehlers-Danlos) can cause AR (due to aortic root dilation)
- 32. **Flame in heart**: infective endocarditis can cause AR (due to valve damage)
- 33. Eccentric myocardial ribbon: AR can cause chronically \uparrow EDV and pressure— LV eccentric hypertrophy
- 34. **Ejecting heart**: eccentric hypertrophy of the LV allows for \uparrow SV to maintain CO
- 35. **Bulging up and down**: \uparrow SV in AR causes \uparrow SBP and \downarrow DBP \rightarrow widened pulse pressure (e.g. 160/60)
- 36. **Hammering water**: AR causes rapid filling and collapse of blood vessels (corrigan or "water hammer" pulse)
- 37. **Bobbing doll head**: AR can cause characteristic head bob with each pulsation (de Musset sign)
- 38. **Jumping on nail bed**: ↑ pulse pressure in AR can cause pulsation in the lips or nailbeds (Quincke pulse)
- 39. **Murmur and diamonds**: murmur of AR is described as blowing, decrescendo diastolic murmur (best appreciated at left sternal border in 3 or 4 intercostal space)
- 40. Sloshing Slushi3: dilation of LV with AR can cause S3 heart sound

Cardiac Pathology: Chapter 5.1 L to R Shunt



- 1. Read comics left to right: left-to-right shunts (e.g. VSD, ASD, PDA)
- 2. **75% to 80% markdown** : left-to-right shunts increase the O2 saturation in the right chambers of the heart
- 3. **Rumbling diamond cave**: ASD can present with a diastolic rumble (increased flow across the tricuspid valve)
- 4. **Transforming Dr. Eisenmenger**: Eisenmenger syndrome (left-to-right shunts reverse into right-to-left shunts due to formation of pulmonary artery hypertension
- 5. Tense arterial strings on chest : left-to-right shunts can cause pulmonary artery hypertension (PAH) \rightarrow Eisenmenger syndrome
- 6. Late blue transformation : left-to-right shunt reversal leads to late onset cyanosis
- 7. Torn open chest ${\bf V}$: ventricular septal defect (VSD a left-to-right shunt)
- 8. **Systolic spray from little V-man**: a small VSD presents with a systolic ejection murmur
- 9. Hologram comic: a small VSD presents with a holosystolic murmur
- 10. Protected baby: a small VSD is usually asymptomatic
- 11. **Falling feeding baby**: a large VSD can cause failure to thrive and diaphoresis with feeding
- 12. Failing heart balloon: a large VSD can cause heart failure
- 13. "Please use second ostium" : the ostium secundum forms in the septum primum $\,$
- 14. **Second set of septal doors**: the septum secundum grows to cover the septum primum (and ostium secundum)
- 15. Holding open septal doors : incomplete formation of the septum secundum leaves an opening that communicates with the ostium secundum \rightarrow atrial septal defect (ASD)
- 16. "Incomplete fusion": incomplete fusion of the septum primum with the septum secundum leaves a patent foramen ovale (PFO)
- 17. Shooting through the septal doors from right to left: reversal of flow through an ASD or PFO allows a "paradoxical embolism" to reach the systemic circulation
- 18. **Paradoxical dart hitting head** : reversal of flow through an ASD or PFO can cause cryptogenic stroke (due to paradoxical embolism)
- 19. **Straining to hold septal doors open**: valsalva can reverse flow through an ASD or PFO (right to left shunt)
- 20. Shooting through the septal doors from right to left: reversal of flow through an ASD or PFO allows a "paradoxical embolism" to reach the systemic circulation
- 21. Systolic spray: ASD can present with a systolic murmur
- 22. **Split sisters**: ASD can present with a wide fixed splitting of the S2 heart sound

- 23. **Bubbles floating through septal doors**: ASD has a positive bubble study on cardiac echo
- 24. **Open air ductus** : patent ductus arteriosus (PDA - a left-to-right shunt)
- 25. **Ruby robot opening air ductus** : congenital rubella infection can present with a PDA
- 26. **Pro slugger bat opening air ductus**: prostaglandin E2 (produced by the placenta) keeps the ductus arteriosus open
- 27. Fire extinguisher fending off baseball bat kid: indomethacin (an NSAID) closes a PDA (blocks prostaglandin production)
- 28. Murmur from air conditioner machine: PDA can present with a continuous "machine-like" murmur (present during systole and diastole)
- 29. **Red body, blue legs**: an untreated PDA (leading to Eisenmenger's) can cause cyanosis in lower extremities
- 30. Colorful arch: coarctation of the aorta
- 31. **Dent near the air duct**: coarctation of the aorta causes a narrowing of the descending aorta near the insertion of the ductus arteriosus
- 32. **Turning X shaped pinwheel** : coarctation of the aorta can occur with Turner syndrome (monosomy X)
- 33. **Bicuspid horse mout**h : coarctation of the aorta can be associated with a bicuspid aortic valve
- 34. **Pink body, blue legs**: severe coarctation of the aorta can cause cyanosis in lower extremities (presents in infancy)
- 35. **Failing heart balloon**: severe coarctation of the aorta can cause heart failure and shock (after closure of PDA)
- 36. **High pressure steam** : coarctation of the aorta can cause hypertension
- 37. **Split pi**pe : coarctation of the aorta can cause aortic dissection
- 38. **Red paint st**roke: coarctation of the aorta can cause hemorrhagic stroke (due to ruptured berry aneurysm)
- 39. **High pressure in upper half of pipe**: coarctation of the aorta can cause a blood pressure discrepancy between the upper and lower extremities (or rarely between arms)
- 40. **Delayed events**: coarctation of the aorta can cause delayed lower extremity pulses ("brachial-femoral delay")
- 41. **Paint clods on l**egs: coarctation of the aorta can cause lower extremity claudication
- 42. **Notched rungs**: coarctation of the aorta can cause inferior "rib notching" on CXR (due to collateral circulation in the intercostal arteries)
- 43. Flame in heart lantern: left-to-right shunts (e.g. VSD, PDA, coarctation) can cause endocarditis
- 44. **Destroying endocarditis monster**: ASD is unlikely to cause endocarditis (low pressure differential between the atria causes less hemodynamic injury)



- Read manga right to left: right-to-left shunts (e.g. truncus arteriosus, TGV, tricuspid atresia, TOF, TAPVR)
- 2. Blue baby: right-to-left shunts cause early onset cyanosis
- 3. Large purple trunk: truncus arteriosus (right-to-left shunt)
- 4. Large V shirt: truncus arteriosus often occurs with VSD
- "September" :failure of septation causes truncus arteriosus (no division between aorta and pulmonary trunk)
- Neural crest shield: failure of neural crest cell migration causes truncus arteriosus
- 7. **"22" and "11"** helmet: failure of neural crest cell migration (e.g. truncus arteriosus) is associated with 22q11 deletion syndromes (e.g. DiGeorge)
- 8. **Monster trainer station**: transposition of the great vessels (TGV a right-to-left shunt)
- Independent right and left circuits: TGV results in an independent deoxygenated systemic circuit and oxygenated pulmonary circuit (aorta and pulmonary artery are transposed)
- 10. "PDA VS ASD": TGV is incompatible with life without a VSD, ASD, or PDA (left-to-right shunts)
- 11. **Murmur between the circuits**: TGV can present with a murmur form an associated left-to-right shunt
- 12. **Red ponytail in front of blue**: with TGV, the aorta is anterior to the pulmonary artery (on cardiac echo)
- Monster ball on a string :with TGV, the cardiac silhouette looks like an "egg on a string" on CXR
- 14. Throwing up candy: diabetes in the mother is a risk factor for TGV
- Defeated spiral monster: failure of the aorticopulmonary septum to spiral results in TGV
- 16. Tricuspid "Z" warrior: tricuspid atresia (right-to-left shunt)
- Hole in septal window: tricuspid atresia is often associated with an ASD
- 18. "Tetra Sailors": tetralogy of Fallot (Right-t-left shunt)
- Earth sailor with constricted pulmonary trees: pulmonary valve stenosis (feature of TOF)
- Earth sailor's blue face: pulmonary valve stenosis causes cyanosis (the degree of stenosis dictates the severity of disease)
- 21. Water sailor's conch shell: TOF is associated with right ventricular hypertrophy (concentric) due to pressure overload

- 22. Water sailor's large boots: right ventricular hypertrophy (feature of TOF) seen on CXR as a "boot-shaped heart"
- 23. Fire sailor's big V neck: ventricular septal defect (feature of TOF)
- 24. Air sailor's red ponytail flying overhead: overriding aorta (feature of TOF)
- 25. Neural crest shield: failure of neural crest cell migration causes TOF
- 26. "22" and "11" jewelry: failure of neural crest cell migration (e.g. TOF) is associated with 22q11 deletion syndromes (e.g. DiGeorge)
- 27. **Systolic spray**: TOF can present with a harsh systolic crescendo/decrescendo murmur (due to pulmonary valve stenosis)
- Evil choking spell: TOF can present with hypercapnic spells ("tet spells")
- 29. **Squatting to dodge spell**: squatting relieves symptoms during a hypercapnic spell
- 30. **Geyser shooting upward**: squatting increases SVR forcing more blood upward into the pulmonary circulation
- 31. "Tap VR": total anomalous pulmonary venous return (TAPVR right-to-left shunt)
- 32. **Right, down, right, down...**: in TAPVR, the pulmonary veins drain back into the right heart (with the systemic circulation)
- 33. **Dilated right tap dancer**: TAPVR causes a dilated right atrium and ventricle
- 34. **Hole in septal window**: in TAPVR, an ASD allows some oxygenated to enter the systemic circulation
- 35. "Upstairs": Ebstein's anomaly can present in infancy with cyanosis
- 36. Large atrium map: Ebstein's anomaly is associated with dilation of the right atrium
- 37. "Event in atrium": the abnormal dilation of the right atrium and inferior displacement of the tricuspid valve into the ventricle istermed "atrialization" of the right ventricle
- 38. **Regurgitation on tricuspid wig**: Ebstein's anomaly is associated with a malformed tricuspid valve and tricuspid regurg
- 39. **Failing heart balloon**: Ebstein's anomaly is associated with right sided heart failure (due to severe tricuspid regurg)
- 40. **Pregnant mother on "lift-ium":** Ebstein's anomaly is caused by lithium exposure in utero



- Obstructing street: in COPD, obstruction prevents air from leaving the lungs (trapped in distal airways)
- 2. **Collapsed in the terminal street**: collapse at the distal terminal bronchioles causes air trapping
- 3. "NO U TURN": COPD causes irreversible obstruction
- 4. Smoker: cigarette smoking is the most important risk factor for COPD
- Pink puffer in distal cul-de-sac: emphysema (permanent airway dilation) affects the distal airways
- 6. End of cul-de-sac : alveolar sac
- 7. Distal cul-de-sac path: alveolar duct
- 8. Proximal cul-de-sac path: respiratory bronchiole
- 9. **Blue bloater in the proximal street**: chronic bronchitis (chronic productive cough) involves the proximal large airways
- "Road Terminates": terminal bronchioles (emphysema occurs distally, while chronic bronchitis involves the airways here and more proximally)
- 11. **Dead grass at cul-de-sac entry**: centriacinar emphysema affects respiratory bronchioles and spares alveolar ducts and sacs
- 12. **Sweaty upper jersey**: centriacinar emphysema preferentially affects the upper two-thirds of the lung
- 13. **Toxic hockey puck**: toxins (e.g. in cigarette smoke) collect in the respiratory bronchioles and activate an inflammatory response
- 14. First responders: neutrophils recruited to distal airways
- 15. First responder cutting elastic bandage: neutrophils produce elastase (breaks down elastin)
- Raised COMPLIANCE book: damage by elastin increases compliance of the distal airway (decreased elasticity)
- 17. **AA trimming**: alpha-1 antitrypsin (major serum inhibitor of neutrophil elastase)
- 18. **Uninhibited shears** : AAT deficiency → uninhibited neutrophil elastase → destruction of distal airways
- 19. Clippings throughout cul-de-sac: AAT deficiency causes panacinar emphysema (affects entire respiratory unit)
- 20. **Torn shirt bottoms**: AAT deficiency predominantly affects the lower lobes of the lung
- 21. **Collections in liver bush**: AAT deficiency → non-secreted AAT accumulates in hepatocytes → liver damage and cirrhosis
- 22. "PASS" frisbee: non-secreted AAT stains PAS positive

- 23. **Young trimmer**: AAT deficiency causes emphysema in young patients
- 24. Smoking: smoking increases emphysema risk in patients with AAT deficiency (directly inhibits AAT)
- 25. **Huffing and puffing**: emphysema presents with gradually progressive dyspnea
- 26. Party blower: emphysema presents with diffuse bilateral wheezing
- 27. Tripod position : classic emphysema pose (extra anchor for accessory muscles of respiration)
- Pursed lips: helps maintain pressure to inflate distal airways (in emphysema)
- 29. Skinny arm: emphysema may cause weight loss
- 30. Inflated chest kite: emphysema causes hyperinflated lungs (on CXR: flat diaphragm, 10+ posterior ribs shadows, increased parenchymal radiolucency, lengthened cardiac silhouette)
- 31. "Paradoxus": emphysema can cause pulsus paradoxus (causes a > 10 mmHg decrease in systolic pressure during inspiration)
- 32. **Distant heart kites**: emphysema presents with distant heart sounds
- 33. **Distant lung sails**: emphysema can present with diminished lung sounds
- 34. Full "Total Load" bin : COPD causes increased total lung capacity (TLC)
- 35. Full "Residual" bin : COPD causes increased functional residual capacity (FRC)f
- 36. **Dropping "ForEVer #1" sign**: COPD causes decreased forced expiratory volume in 1 second (FEV1)
- 37. **Dropping "ForeVer Champs" sign** : COPD causes decreased forced vital capacity (FVC)
- 38. 7-shaped hockey stick: FEV1/FVC is < 0.7 in COPD
- 39. Colorless heme plastic trash LOW on the ground: emphysema causes a low DLCO (diffusion capacity of the lung for carbon monoxide)
- 40. **Pink face**: hyperventilation EARLY in course maintains normal arterial oxygen level (PaO2)
- 41. **Blowing OH bubbles**: hyperventilation EARLY in course causes respiratory alkalosis
- 42. **Popping OH bubbles**: in LATE emphysema there is severe air trapping → CO2 retention and respiratory ACIDOSIS
- 43. Late blue face : in LATE emphysema there is a severe decrease in DLCO \rightarrow decreased PaO2 (hypoxemia) \rightarrow cyanosis



- 44. **Blue bloater #32**: chronic bronchitis is defined as productive cough for at least 3 consecutive months in each of 2 consecutive years
- 45. **Hacking up sports drink**: chronic bronchitis presents with a productive cough
- 46. **Mucus on tracheal stick**: chronic bronchitis involves mucus gland hypertrophy and hypersecretion in larger airways (trachea, bronchi, and bronchioles)
- 47. **Goblet bottles in terminal street**: chronic bronchiolitis (as part of chronic bronchitis) causes goblet cell metaplasia and proliferation
- 48. **Bottle plugs**: mucus hypersecretion causes mucus plugs in bronchioles → distal airway obstruction (in chronic bronchitis)
- 49. **CO2 fumes** : EARLY in course mucus plugs trap air in distal airways → increased PaCO2 and respiratory ACIDOSIS (in chronic bronchitis)

- 50. **Blue face**: EARLY in course mucus plugs limit oxygenation of alveoli → decreased arterial pO2 (hypoxemia) → cyanosis (in chronic bronchitis)
- 51. **O2 knocking over rate**: O2 supplementation can decrease RR causing respiratory failure in COPD patients
- 52. **O2 knocking over arch**: O2 supplementation inhibits firing of peripheral chemoreceptors (aortic arch and carotid bodies sense decrease in PaO2)
- 53. **Hypoxic goalie stretching net** : chronic hypoxemia in COPD → hypoxic vasoconstriction → pulmonary arterial hypertension
- 54. Corked heart bottle : pulmonary hypertension due to hypoxic vasoconstriction in COPD can lead to right heart failure (COR PULMONALE)



- 1-Hyperreactive to bee : asthma is characterized by a hyperreactive airway
- 2-Backwards cap: asthma, unlike emphysema, is characterized by REVERSIBLE, intermittent airway inflammation and obstructive symptoms
- 3-**Obstructing exit**: in asthma, obstruction prevents air from leaving the lungs (trapped in distal airways)
- 4-Flame and slingshot: asthma is characterized by chronic bronchial inflammation (with eosinophils)
- 5-Mucous dripping : asthma is characterized by increased mucus secretion
- 6-**Grip on limb**: asthma is characterized by smooth muscle hypertrophy and hyperreactivity
- 7-Green distal branches: inflammation, mucus secretion, and smooth muscle hypertrophy confined to larger airways (respiratory units spared)
- 8-Antigenic squirrel: atopic asthma involves a type I hypersensitivity reaction, with common triggers being animal dander, pollen, dust, and other environmental antigens
- 9-IgE archer aiming at beehive: in response to antigen, helper T cells release IL 4 and 13 signal B cells to produce IgE (IL 4 and 13)
- 10-**Testing air with 1 finger**: the inflammatory reaction in asthma is a type 1 hypersensitivity reaction (IgE production and mast cell degranulation)
- 11-Bee hive : mast cell (inflammatory reaction in asthma initiated by mast cell degranulation Type I HSR)
- 12-Arrows coating bee hive : IgE produced by B cells coat mast cells throughout the large airways
- 13-Squirrel crosslinking arrows : the re-introduced antigen crosslinks lgE on mast cells \rightarrow degranulation
- 14-Bee release: degranulation of mast cells releases histamine, acetylcholine, leukotrienes, and other proinflammatory molecules
- 15-**Gripping, drooling, dilated sleeves**: proinflammatory molecules released by mast cells induce bronchoconstriction, mucus production, and vasodilation in large airways (EARLY PHASE of acute asthma exacerbation)
- 16-Late flame cape: inflammation consisting of eosinophils, neutrophils, and T cells occurs 4-8 hours after the early phase (LATE PHASE of acute asthma exacerbation)

- 17-Late slingshot kid : a characteristic finding in asthma is eosinophilic inflammation
- 18-Damaged major base: a major source of damage from eosinophilic inflammation is release of major basic protein, an anthelmintic toxin that causes epithelial damage, histamine release, and further eosinophil chemotaxis
- 19-"Remodeling": chronic inflammation from repeated attacks causes permanent structural changes to the bronchial wall (airway remodeling)
- 20-**Chronic flame cape** : recurrent, chronic INFLAMMATION leads to airway remodeling
- 21-Buff kid : airway remodeling in chronic asthma includes bronchial smooth muscle hypertrophy
- 22-Thick concrete base: airway remodeling in chronic asthma includes thickened basement membrane
- 23-**Mucousy water bottles** : airway remodeling in chronic asthma include hypertrophy of submucosal mucus glands
- 24-Pink granules in puddle: eosinophilic infiltration of the airways is one of the hallmarks of atopic asthma (eosinophils in sputum and eosinophilia)
- 25-Can plugged with mucus: mucus plugs in bronchi and bronchioles
- 26-Curly string from plugged can: sloughing of the bronchial epithelium can lead to formation of Kurshmann spirals, which are whorled deposits of epithelial cells
- 27-Pink jacks : Charco-Layden crystals are thin, needle-like concretions of eosinophilic proteins seen in the sputum of asthmatics



- 28-Kid puffing air out : acute dyspnea is a common symptom of asthma exacerbation
- 29-**Kid waking up coughing**: chronic cough, especial nocturnal cough, in children may be the only presenting symptom of asthma
- 30-**Family photo**: asthma is highly associated with atopy, so a family history of asthma or allergies is common
- 31-Billowing lungs: air trapping in acute exacerbations can be seen on chest x-ray as hyperinflation (flattened diaphragms, and lengthening of the cardiac silhouette)
- 32-PULSUS PARADOXUS: severe asthma attacks can lead to pulsus paradoxus, a drop in systolic BP >10mmHg on inspiration
- 33-Falling FEV1/FVC signs : classic spirometry findings in asthma are a FEV1/FVC ratio <0.7 and an FEV1 <80% expected
- 34-Blowing OH bubbles: patients with acute asthma exacerbations will have an initial respiratory alkalosis from hyperventilation (can progress to acidosis as severity increases)
- 35-Viral lantern: viral infections are a common inciting cause of nonatopic asthma exacerbations
- 36-Ashtray: second hand smoke and air pollutants are another common cause of non-atopic asthma
- 37-**Umpire holding lacrosse stick**: aspirin is the most common cause of drug induced asthma (inhibiting COX-1 shifts AA metabolism to LOX pathway → leukotrienes → bronchoconstriction)
- 38-Inflamed dilated pomegranates: bronchiectasis is characterized by permanent dilation of the bronchi and bronchioles (due to infections and inflammation that destroy the muscle and elastic tissue supporting the airway
- 39-Recurring bacterial lanterns : bronchiectasis is caused by chronic recurrent bacterial infections
- 40-**Crab drawing**: tumors causing obstruction can lead to distal infection, thus initiating the cycle of infection/inflammation \rightarrow bronchiectasis
- 41-**Thick tree sap**: CF is the most common cause of bronchiectasis in the US (thick secretions cause obstruction leading to infection/inflammation)

- 42**Broken ciliated moss**: primary cilia dyskinesia is another possible cause of bronchiectasis (secretions are not cleared due to dysfunctional cilia)
- 43-Upper lobe holes: tuberculosis is the most common cause of bronchiectasis worldwide
- 44-**Crowded lower pomegranates**: bronchiectasis is primarily affects lower lobes (seen on CXR and CT as CROWDED bronchial markings extending to the edge of the lung periphery
- 45-Rotten pomegranate: bronchiectasis is characterized by copious sputum production, often describes as "cup fulls"
- 46-Juicy red pomegranate: extensive parenchymal damage and inflammation can lead to hemoptysis in bronchiectasis

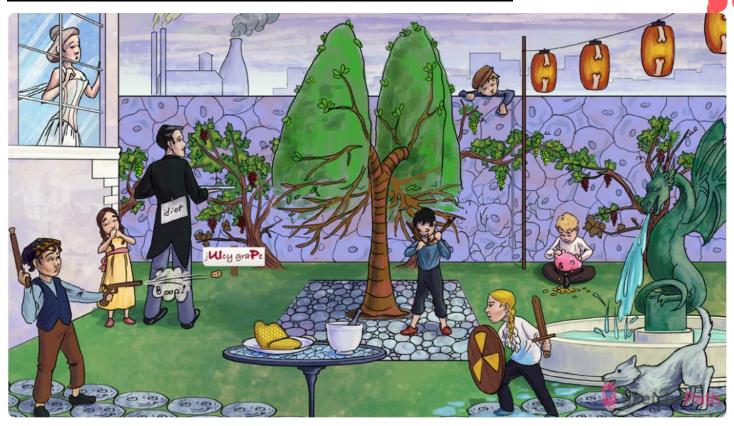
Restrictive lung disease (Overview)



- Torn compliance contract: lung compliance decreased in restrictive lung disease
- Total "Load" Capacity overturned : total lung capacity (TLC) decreased in restrictive lung disease. Therefore unable to take a very large breath
- Elevated FEV1/FVC signs: Forced Expiratory Volume in 1sec / Forced Expiratory Volume (FEV1/FVC) is elevated (>80%) in restrictive lung disease
- Falling FVC sign: FVC decreases in restrictive lung disease (FEV1/FVC increases)
- FEV1 banner pulled tight: increased elasticity of pulmonary interstitium (interstitial restrictive lung disease) → airway widening and decreased resistance to expiratory flow → maintains FEV1 (though still decreased)
- Overturned residual capacity: Functional Residual Capacity (FRC) is decreased in restrictive lung disease.
- Restrictive corset cough: non-productive cough in interstitial lung disease (INTRINSIC restrictive lung disease)
- 8. Over-exerted breath: restrictive lung disease starts with dyspnea on exertion and can progress to dyspnea at rest
- Ripping corset velcro straps: interstitial lung disease (INTRINSIC restrictive lung disease) can cause dry crackles ("velcro rales") usually heard best at the lung bases.
- 10. **X jolly roger**: interstitial lung disease (INTRINSIC restrictive lung disease) can be seen on x-ray (diffuse reticulo-nodular opacities)
- 11. **Reticular knotted pattern**: interstitial lung disease (INTRINSIC restrictive lung disease) commonly presents with reticulo-nodular, diffuse, and bilateral opacities on x-ray.
- 12. **Tight red corset ribbons**: chronic interstitial lung disease can cause pulmonary hypertension (destruction of lung parenchyma and reduction in alveolar capillaries → increased pulmonary arterial resistance)
- Corked bottle with heart ship: Pulmonary hypertension can cause right heart failure (COR PULMONALE)
- 14. **Pleural shirt**: Pleural diseases (e.g. mesothelioma) and pleural effusions can cause EXTRINSIC restrictive lung disease
- 15. Muscles and cut communication wire: neuromuscular diseases (e.g. polio or myasthenia gravis) can cause EXTRINSIC restrictive lung disease when diaphragmatic and intercostal muscles affected

- 16. Locked chest of drawers: Spine malpositioning (e.g. kyphoscoliosis, ankylosing spondylitis) can restrict chest wall expansion and cause EXTRINSIC restrictive lung disease
- 17. **Obese Governor Pickwick**: Obesity can limit chest wall expansion and cause EXTRINSIC restrictive lung disease
- 18. **Shallow breathing into bag**: obese patients may take faster smaller breaths due to extrathoracic restriction (retention of carbon dioxide)
- Low extra reserves: the most common indicator of obesity-related restrictive lung disease is a reduction in Expiratory Reserve Volume (ERV)
- 20. **Hypoxic blue face**: obese patients may develop chronic restrictive lung disease → retention of carbon dioxide (Obesity Hypoventilation Syndrome) with high PaCO2 and low PaO2
- 21. **Tight vascular vest chains** : obesity can cause chronic hypoxia → chronic pulmonary vascular constriction→ pulmonary hypertension
- 22. Corked bottle with heart ship : pulmonary hypertension caused by obesity-related restrictive lung disease can lead to right heart failure (COR PULMONALE)
- 23. **fibrotic pulmonary trees**: idiopathic pulmonary fibrosis (INTRINSIC restrictive lung disease)
- 24. **Dusty factory**: pneumoconioses (INTRINSIC restrictive lung disease)
- 25. Soccer player: sarcoidosis (INTRINSIC restrictive lung disease)
- 26. Odorless colorless plastic trash littered on ground: DLCO is LOW in INTRINSIC restrictive lung disease only (e.g. pulmonary fibrosis, pneumoconiosis) because diffusion surface is destroyed
- 27. **Ground glass mirror**: reticulo-nodular opacities may be described as "ground glass"

Idiopathic Pulmonary Fibrosis (IPF)



- Restrictive corset: interstitial lung diseases (e.g. idiopathic pulmonary fibrosis (IPF) produce restrictive lung disease
- Fibrotic pulmonary tree: pulmonary fibrosis (a component of many of the interstitial lung diseases)
- 3. "Idiot": Idiopathic pulmonary fibrosis (IPF) is the prototypical fibrosing disorder
- 4. Repeating red grapes: IPF is associated with repeated cycles of alveolitis (of unknown origin)
- 5. Cracks in epithelial stones: recurring inflammation damages type 1 and type 2 alveolar cells in the alveolar epithelium
- 6. **Dumping coins** : damaged type-1 pneumocytes release cytokines → TGF-beta-1 activates fibroblasts → pulmonary fibrosis
- 7. **Patchy distribution of grapevines**: IPF is associated with a patchy fibrosis (due multiple fibroblastic foci) on histology
- 8. "jUlcy graPe" : usual Interstitial pneumonia (UIP) is the patchy fibrotic histology seen in IPF
- 9. **Cobblestone patio**: IPF is associated with a cobblestone appearance of the pleural surface (retraction scars along the interlobular septa)
- Bare lower branches: fibrotic changes in IPF appear as bilateral or diffuse reticular opacities, most prominent in LOWER LOBES (on X-ray or CT)
- 11. **Branches under shirt**: the opacities of IPF distribute along SUB-PLURAL regions and interlobular septa
- 12. **Honeycomb treat**: alveoli collapse and dilated proximal airways in IPF appear as "honeycombing" on CT and gross pathology
- 13. **CAP gun going "BOOP"**: cryptogenic organizing pneumonia (COP) also known as bronchiolitis obliterans organizing pneumonia (BOOP) is another cause of pulmonary fibrosis
- 14. Plug in gun: COP is associated with intraluminal plugs of granulation tissue leading to alveolar collapse and consolidation →alveolar collapse and consolidation
- 15. Sudden gunfire: COP causes acute onset of cough and dyspnea
- 16. Fire bandana: COP presents with fever and weight loss
- 17. Moon face: COP can be treated with oral corticosteroids
- 18. **Mortar and pestle**: many drugs (e.g. amiodarone, bleomycin, methotrexate) can cause pulmonary fibrosis

- 19. **Fibrous radiation shield**: patients with history of thoracic radiation can develop radiation pneumonitis and pulmonary fibrosis
- 20. Wet pleural shirt: radiation pneumonitis can present with pleural effusion
- 21. **Moon face**: radiation pneumonitis can be treated with oral corticosteroids
- Lupus wolf: collagen vascular diseases (e.g. lupus) can cause pulmonary fibrosis
- 23. Scaly dragon: systemic sclerosis can cause pulmonary fibrosis
- 24. **Inflamed joint lanterns**: rheumatoid arthritis can cause pulmonary fibrosis



- 1. **Particulates in air**: pneumoconioses are interstitial lung diseases caused by the inhalation of organic and inorganic particulates
- Restrictive corset: pneumoconioses can present with a restrictive lung disease picture (reduced lung compliance, FEV1, FVC, and TLC)
- Screw with nuts: in the macrophages, asbestos fibers are coated with an iron containing proteinaceous material → ferruginous bodies (brown "beaded appearance" on H&E)
- 4. Larger particles on belt : larger particles (10-15 microns) will get trapped in upper airway
- Sweeping medium particles: particles 5-10 microns in diameter are cleared by mucociliary transport in the trachea and bronchi
- Small particles trapped at bifurcations: particles 1-5 microns in diameter lodge at the bifurcation of respiratory bronchioles → phagocytosed by macrophages
- 7. **Small particles in cages**: particles 1-5 microns in diameter are engulfed by alveolar macrophages → cytokine release
- 8. **Dropping coins**: cytokines (PDGF, IGF) released from macrophages are the cause of inflammation and fibrosis in pneumoconioses
- 9. **Shark tattoo**: collagen production from the release of growth factors leads to pulmonary fibrosis and restrictive lung disease
- 10. **Cigar**: tobacco smoke worsens symptoms and clinical course of all the pneumoconioses
- 11. Black panther coal: pulmonary anthracosis consists of asymptomatic pigment deposition in interstitial tissue and hilar nodes (contained in macrophage "dust-cells")
- 12. **Streaked black sails**: streaks of anthracotic pigment are seen throughout the lungs (lymphatic spread of "dust cells")
- 13. **Hilar coal cages** : anthracotic pigment is deposited in the hilar lymph nodes (lymphatic spread of "dust cells")
- 14. **Coal on lung coral**: simple CWP is characterized by "coal macules" and focal fibrotic "coal nodules" (predominantly in the upper lobes)
- 15. X-ray flag: simple CWP shows small, rounded, opacities, in the upper
- Puffer fish in center: simple CWP produces centriacinar emphysema (mostly in the upper lobes)

- 17. Bigger chunks on lung coral : Complicated CWP is characterized by massive blackened opacities and fibrosis (predominantly in the upper lobes)
- Sandblaster: exposure to silica occurs in foundries, mines, sandblasting (quartz is particularly fibrogenic)
- 19. Sand crystals on lung coral: silicotic nodules are found mostly in the upper lung fields
- 20. Whorled shell: silicotic nodules contain concentrically arranged collagen
- 21. Fragrance from whorled shell: silicotic nodule will appear as weakly birefringent particles under polarized light
- 22. **Honeycomb pattern**: nodules coalesce to form large scars with areas of honeycombing in between (cystically dilated)
- 23. **Hilar shells**: silicosis causes "egg-shell" calcification of the hilar lymph nodes (fibrosed lymph nodes)
- 24. **Cowboy breaking cage**: silicosis increases risk of TB infection (disrupt phagolysosome and promote apoptosis)
- 25. **Big rust holes**: In the setting of a pulmonary TB infection, nodules of silicotuberculosis can form, containing a central zone of cassation
- 26. Pink insulation : asbestos exposure can cause asbestosis: a pneumoconiosis characterized by slow progressive and diffuse pulmonary fibrosis)
- 27. Ship builder: asbestos can be found on ship plumbing insulation, ceiling tiles and floor tiles
- 28. **Nails and screws**: asbestos fibers may be straight, stiff, and brittle (amphibole) or curly and flexible (serpentine)
- 29. **Straight nail in shirt**: amphibole fibers can penetrate the epithelium and enter the interstitium (more pathogenic than "serpentine")
- Lower barnacles: the fibrosis of asbestosis predominantly affects the subpleural lower lung fields
- 31. **Large buttons**: pleural plaque formation is the most common manifestation of asbestos exposure (benign, no asbestos bodies)
- 32. **Honeycomb shape**: in asbestosis, fibrosis progresses to large inelastic fibrous tissue segments with intervening areas of "honeycombing"



- Soccer ball: sarcoidosis (a multisystem granulomatous disease with major pulmonary findings)
- Intact macro-CAGES: sarcoidosis is associated with non-caseating granulomas (a collection of macrophages without an area of central necrosis)
- 3. Black female soccer captain: sarcoidosis is most common in African Americans (particularly young females between 20-39)
- 4. No smoking sign: sarcoidosis is more common in non-smokers
- 5. Helper T squires: CD4+ helper T-cells are activated in Sarcoidosis
- "BAL" bottle: bronchoalveolar lavage shows an elevated CD4+ to CD8+ ratio (> 2:1) in sarcoidosis
- No reaction to feather: sarcoidosis can cause anergy to common skin antigens that usually elicit type-IV (delayed) immune reactions (e.g. Candida, PPD test)
- Antibody keys: sarcoidosis can cause polyclonal hypergammaglobulinemia (due to Helper T cell dysregulation)
- 9. **Multiple purple panels**: granulomas may contain multinucleated giant cells (formed by the fusion of activated macrophages)
- 10. **Ball with star panels**: giant cells may contain asteroid bodies (stellate inclusions)
- 11. **Show-man with purple cleat**: granulomas may contain Schaumann bodies that show up as a purple spot on histology
- 12. Calcified leather cleat: Schaumann bodies contain laminated calcium and protein
- 13. **Balls in the field**: non-caseating granulomas can be found throughout the lung interstitium in sarcoidosis
- 14. **Soccer balls at the midline**: non-caseating granulomas can occur in hilar and paratracheal lymph nodes → hilar lymphadenopahty
- 15. **Hilar soccer balls in lung tree**: in sarcoidosis, enlarged bilateral hilar and mediastinal lymph nodes can be seen on chest x-ray
- 16. Fibrotic lung tree : in sarcoidosis, pulmonary granulomas can be replaced by diffuse interstitial fibrosis
- 17. **Dyspneic player**: pulmonary sarcoidosis presents with a gradual onset of dyspnea (on exertion)
- 18. Coughing player: pulmonary sarcoidosis can present with a dry cough

- 19. **Skinny goalie with flame bandana**: sarcoidosis presents with other constitutional symptoms (malaise, fever, anorexia, weight loss)
- 20. Painful spotted shin guards: sarcoidosis can present with erythema nodosum (raised red painful nodules on anterior legs; no granulomas)
- 21. **Gravel nodules**: sarcoidosis can present with subcutaneous nodules (non-painful; contain abundant granulomas)
- 22. **Purple face paint**: sarcoidosis can cause lupus pernio (violaceous rash on nose and cheeks)
- 23. **Blurry red rimmed goggles**: sarcoidosis can cause anterior uveitis → redness, blurry vision, glaucoma
- 24. **Retina street lights with broken wires** : sarcoidosis can present with retinal and optic nerve involvement → vision loss
- 25. **Dry water bottle**: sarcoidosis can present with lacrimal and salivary gland involvement → dry eye and dry mouth
- 26. Liver spot cow : sarcoidosis can involve the liver \rightarrow granulomatous hepatitis
- 27. Restrictive net : cardiac sarcoidosis may cause restrictive cardiomyopathy
- 28. **Raised milk glass**: Sarcoidosis can cause hypercalcemia (due to hypervitaminosis D)
- 1-α Box : activated macrophages in granulomas produce 1-αhydroxylase (converts Vitamin D into its active form, 1-25dihydroxyvitamin D)
- 30. **Sunny street lights**: extra 1-î±-hydroxylase produced in the granulomas may lead to hypervitaminosis D → hypercalcemia
- 31. **Stones in leaked milk** : sarcoidosis can present with hypercalciuria → calcium kidney stones
- Raised ACE card: sarcoidosis can present with increased levels of angiotensin converting enzyme (ACE) (produced in the granulomas)
- 33. **Moon face balls**: progressive sarcoidosis can be treated with glucocorticoids
- 34. **Building aircraft**: beryllium dust is found in nuclear and aerospace industries (exposure can lead to berylliosis)
- 35. **Macro-CAGES** with soccer ball: berylliosis presents with non-caseating granulomas (similar to sarcoidosis)
- 36. Particles falling on top of fibrotic lung tree: interstitial fibrosis in berylliosis may be more prominent in upper lobes



- Broken vacuum: a pneumothorax results in the loss of negative intrapleural pressure
- Huffing and puffing: pneumothorax presents with the sudden onset of dyspnea
- 3. **Sharp necklace**: pneumothorax causes pleuritic chest pain on the affected side (SHARP pain provoked by deep inspiration)
- 4. **Resonant drum**: pneumothorax presents with hyperresonant percussion on the affected side
- Distant lung sails: pneumothorax presents with diminished breath sounds on auscultation of the affected side (due to the air filling the pleural space)
- Tall thin healthy guy: primary spontaneous pneumothorax occurs most frequently occurs in tall, thin males in their early 20s (no underlying lung disease, no precipitating event)
- 7. **Smoking**: the risk of primary spontaneous pneumothorax increases with smoking
- 8. **Bubbling oil**: primary spontaneous pneumothorax is associated with the formation of apical subpleural blebs
- 9. **Tearing apex**: in primary spontaneous pneumothorax, subpleural arise in the APEX of the lung
- 10. **Blue bloater and pink puffer**: parenchymal destruction in COPD is the most common cause of secondary spontaneous pneumothorax
- 11. **Bubbling to the top**: COPD is associated with the formation of apical subpleural blebs
- 12. **Cancer crabs**: parenchymal destruction from malignancy may lead to secondary spontaneous pneumothorax
- 13. **Pointy cactus**: M. tuberculosis infection can cause pleural or bronchial destruction leading to secondary spontaneous pneumothorax
- 14. Rusty chest plate: pulmonary infections (e.g. pneumonia and lung abscess) can cause secondary spontaneous pneumothorax
- 15. Forcing in air: high airway pressures from mechanical ventilation can cause barotrauma and pneumothorax
- Pointy chest spikes: penetrating trauma to chest or neck (e.g. gunshot wound, knife wound) can cause pneumothorax
- Blunt club: blunt chest trauma can cause rib fracture and pneumothorax

- Spiky collar: neck trauma or iatrogenic pleural damage from improper catheter insertion (jugular or subclavicular) can cause pneumothorax
- 19. White edge: on chest x-ray, look for white visceral pleural line in the lung field (edge of collapsed lung)
- 20. **Collapsed white net**: on chest x-ray, look for the absence of vessel markings in the periphery
- 21. Flap-like tear in sail: tension pneumothorax develops when air enters the pleural space through a flap-like tear in the visceral pleura → air enters but cannot exit → progressive enlargement of the pneumothorax
- 22. **Tension on mediastinal mast**: tension pneumothorax causes compression of mediastinal structures
- 23. **Shifting mast**: on imaging, look for shifting mediastinal structures (e.g. tracheal deviation AWAY from affected lung)
- 24. **Dilated jug**: tension pneumothorax can cause decreased venous return and obstruction of the SVC → jugular vein distension (JVD)
- 25. **Raised heart clock**: tension pneumothorax can cause decreased venous return → hypotension and reflex TACHYCARDIA
- 26. **Emergency needle**: tension pneumothorax is an emergency requiring immediate needle decompression and subsequent chest tube placement
- 27. **Glass of red liquid**: whole blood leaking into the pleural cavity (e.g. from a ruptured intrathoracic aneurysm) causes a hemothorax
- 28. Clotted ice cubes: a hemothorax may contain blood clots in the fluid collection (less likely with bloody pleural effusion)
- 29. **Glass of turbid milk**: leakage of lymph into the pleural space can cause a collection of chylomicron-rich chylous fluid → chylothorax
- 30. Trident: chylous fluid is rich in triglycerides (>110 mg/dL)
- 31. **Cancer crab**: an intrathoracic malignancy (e.g. lymphoma) can cause a chylous effusion due to obstruction of the thoracic duct
- 32. **Biting milk straw**: trauma to the thoracic duct (e.g. iatrogenic tear during surgery) can cause a chylous effusion
- 33. Feet on drum: hemothorax and chylothorax present with dullness to percussion on the affected side

Respiratory Pathology 3.2 Acute Respiratory Distress Syndrome (ARDS)



- Wet life vest: pulmonary edema (result of increased capillary hydrostatic pressure or capillary injury)
- 2. **High-pressure leaky pipes**: increased hydrostatic pressure causes leakage of fluid through pulmonary capillaries → pulmonary edema
- Failing heart balloon: left-sided heart failure causes increased pressure in left heart, which is transmitted to pulmonary venous system → increased pulmonary capillary hydrostatic pressure → pulmonary edema
- 4. **TRANSatlantic ship**: a transudate consists of cell poor, protein poor fluid that accumulates due to increased hydrostatic pressure
- Stenotic bicuspid clam : mitral stenosis causes increased left atrial pressure → increased pulmonary capillary pressure → pulmonary edema
- Regurgitating bicuspid clam: acute mitral regurgitation causes increased left atrial pressure → increased pulmonary capillary pressure → pulmonary edema
- 7. **Overflowing tank** : volume overload (e.g. CHF exacerbation, renal failure) causes increased pulmonary venous pressures → pulmonary edema
- Breaking pipes: pulmonary capillary damage (e.g. ARDS) causes leakage of cell and protein rich serum (exudate) → pulmonary edema
- 9. Emergency exit: an exudate consists of cell rich, protein rich fluid resulting from increased capillary permeability
- 10. Great white ARDS shark: acute respiratory distress syndrome (ARDS) is characterized by bilateral pulmonary edema and hypoxia → respiratory failure
- 11. Cracked alveolar abalone: ARDS is caused by diffuse alveolar injury
- 12. "Septic" bag: sepsis is the most common cause of ARDS
- Fish aspirating shark vomit: aspiration of gastric contents can cause pneumonitis, which can lead to ARDS
- 14. Rusty diving chestplate: pneumonia can cause ARDS
- 15. **Traumatic bone** : severe trauma (lung contusion, fat emboli) can cause ARDS
- 16. Pancreatic sea sponge: pancreatitis can cause ARDS (pancreatic enzymes released into bloodstream damage alveolar epithelium)
- 17. "TRALI" car with IV bags: transfusion-related acute lung injury (TRALI) can cause ARDS
- 18. **Cyto-coins** : initial alveolar injury → cytokine release by macrophages (ARDS EXUDATIVE phase)
- 19. First responders damaging epithelial floor : neutrophils release reactive oxygen species and proteases → direct injury to alveolar epithelium (type I and type II pneumocytes) (ARDS EXUDATIVE phase)

- 20. First responders damaging capillary pipes : neutrophils release reactive oxygen species and proteases → direct injury to alveolar capillary endothelium (ARDS EXUDATIVE phase)
- 21. Collapsing alveolar anemones : injury to type II pneumocytes causes decreased surfactant → alveolar collapse (atelectasis) (ARDS EXUDATIVE phase)
- 22. **Bloody steak**: damage to capillary endothelium causes a bloody, cellular, protein rich exudate to fill alveoli (ARDS EXUDATIVE phase)
- 23. Overgrowth of pink algae : hyaline membranes (pink material formed by exudate mixing with cell debris) line the small airways (ARDS EXUDATIVE phase)
- 24. "DELAYED": The clinical manifestations of ARDS appear 6 to 72 hours after the inciting event
- 25. Sputtering bilateral snorkel: ARDS presents with bilateral diffuse crackles
- 26. **Struggling for air**: ARDS presents with dyspnea, tachypnea, and accessory muscle use
- 27. Hypoxic blue face : ARDS presents with hypoxemia with increased A-a gradient
- 28. **O2** tank drifting away: ARDS causes hypoxemia that is not easily corrected with oxygen supplementation
- 29. White life vest: on CXR, ARDS shows bilateral opacities leading to "white out" appearance
- 30. "Help sharks proliferate": the PROLIFERATIVE stage of ARDS occurs after the EXUDATIVE stage
- 31. **Mopping up the damage**: in the PROLIFERATIVE phase of ARDS, the pulmonary edema resolves
- 32. **Repairing epithelial tile**: during the PROLIFERATIVE phase of ARDS, type II pneumocytes replicate → resynthesis of surfactant and differentiation into type I pneumocytes → replenished epithelium
- 33. Cartilaginous sharks: myofibroblasts deposit collagen throughout the lung → chronic pulmonary fibrosis (a complication of ARDS)
- 34. Fibrotic lung coral: ARDS can lead to chronic pulmonary fibrosis
- 35. **Blimp fish**: serum BNP is increased in heart failure (used to distinguish CHF from ARDS)
- Slushi3: S3/S4 gallops are common findings in heart failure (used to distinguish CHF from ARDS)
- 37. **Distended blue jugs**: JVD is elevated in heart failure (used to distinguish CHF from ARDS)
- 38. **Wedge fish** : pulmonary wedge pressure is elevated (≥18 mmHg) in heart failure (rarely used to distinguish CHF from ARDS)

Respiratory Pathology 3.3 Deep Vein Thrombosis & Pulmonary Embolism



- 1. Occlusive barricade: deep vein thrombosis (DVT)
- 2. **3-wheeled Virchow chair**: Virchow's triad (three risk factors for DVT: venous stasis, hypercoagulability, endothelial injury)
- Prolonged chair sitting: venous stasis is a risk factor for DVT (e.g. hospitalization, long plane flight - or heart failure)
- 4. **Stick pile**: hypercoagulability is a risk factor for DVT (cancer, pregnancy, OCPs, factor V Leiden, prothrombin gene mutation, etc)
- Damaged endothelial tiles: endothelial injury is a risk factor for DVT (e.g. surgery, smoking, atherosclerotic plaque)
- 6. Thin vein design: DVTs are most common in the small distal veins of the leg (causes minor lower leg pain and swelling)
- 7. **Proximal jacket flaps**: clinically significant DVTs form in the large more proximal veins (e.g. popliteal, femoral, iliac veins)
- 8. **Flying buttons**: DVTs in large proximal veins are most likely to embolize
- 9. Fiery heat: acute DVT presents with warmth
- 10. Cringing: acute DVT presents with pain
- 11. Swollen pant leg: acute DVT presents with leg swelling and edema
- 12. WELL: Wells score is used to determine if HIGH or LOW risk for DVT (based on risk factors [i.e. venous stasis, hypercoagulability, endothelial injury] and physical exam findings [swelling, edema, pain, etc])
- 13. **Scattered double-"D" twigs**: DVT presents with an elevated D-dimer (a measure of fibrin degradation products [particularly dimerized fibrin])(NOT specific for DVT)
- 14. Bullhorn: DVT is diagnosed with ultrasound of the leg veins
- 15. **Heparin hunter**: anticoagulation (heparin, warfarin, etc.) is the treatment for DVT (and PE)
- 16. **Wire chimney frame**: an inferior vena cava filter (IVC filter; intercepts clots in IVC) can be placed to treat DVT if anticoagulation is contraindicated (e.g. high bleeding risk: GI bleeds, intracranial tumors)

- Grandfather clock : chronic DVT (recurrent DVT or residual thrombus)
- 18. Bulging pipe : post-thrombotic syndrome is a complication of chronic DVT (venous hypertension and valvular incompetence → chronic venous insufficiency)
- 19. **Leaky venous pipe**: chronic venous insufficiency presents with edema
- Peeling red wall: chronic venous insufficiency presents with skin pigmentation and venous stasis dermatitis (skin peeling due to ulcerations)
- Fibrotic weeds; chronic venous insufficiency can cause lipodermatosclerosis (fibrosis of subcutaneous tissue; starts at medial ankle)

Respiratory Pathology 3.3 Deep Vein Thrombosis & Pulmonary Embolism



- 22. **Thrombotic nest disrupting pulmonary branches**: pulmonary embolism (PE) (thromboembolism that has traveled through the venous system → through right heart → lodges in pulmonary vasculature)
- 23. "Rue Iliofemoral": more proximal DVTs (popliteal, femoral, iliac veins) are more likely to lead to PE
- 24. **Big "Vive" over small "republique"**: PE causes INCREASED V-Q ratio (lung distal to the occlusion has decreased perfusion (Q), but normal ventilation (V)
- 25. **Distributing waste away from thrombotic nest**: in a PE, blood that would normally perfuse the obstructed region is redistributed to other lung areas
- 26. Blue faced effigy: PE causes hypoxemia (low PaO2 due to decreased perfusion to occluded lung areas)
- 27. "A" roof towering over "a" effigy: in PE, there is an increased A-a gradient (because Alveolar pO2 is high & arterial pO2 is low)
- 28. **Overwhelmed blowing air**: PE causes HYPERVENTILATION (manifests as tachypnea and dyspnea) (activation of chemoreceptors and pulmonary irritant sensors → activation of respiratory centers)
- 29. **Blowing "OH" bubbles**: PE causes RESPIRATORY ALKALOSIS (hyperventilation → "blowing-off" CO2 → decreased PaCO2 and increased arterial pH)
- 30. **Wedged black hat**: a PE that affects the lung periphery can cause infarction (perfusion from pulmonary arteries is crucial in periphery due to decreased prominence of bronchial arteries)(causes a wedge-shape of necrosis)
- 31. **Shark-tooth necklace**: pulmonary infarcts (secondary to PE) can cause pleuritic chest pain (sharp pain aggravated by inspiration)
- 32. **Bloody rag**: pulmonary infarcts (secondary to PE) can cause hemoptysis (due to capillary damage)
- 33. "Hampton": pulmonary infarcts (secondary to PE) can cause a Hampton's hump on CXR (peripheral wedge shaped opacity)
- 34. "Westermarque": pulmonary infarcts (secondary to PE) can cause a Westermark sign on CXR (hyperlucent [dark] area distal to embolus due to reduced blood flow)

- 35. **Tense vascular chest chains**: severe PE causes increased pulmonary vascular resistance (due to occlusion of pulmonary artery)(causes acute pulmonary hypertension)
- 36. **Horse saddle**: a "saddle embolus" (large PE straddling the pulmonary artery bifurcation [occludes both left and right pulmonary arteries]) can cause hemodynamic collapse
- 37. **CORKed heart bottle**: a large PE can cause COR PULMONALE (RIGHT heart failure secondary to pulmonary hypertension)
- 38. Raised heart watch: a large PE can cause tachycardia (RIGHT heart failure → diminished filling of LEFT ventricle → decreased cardiac output → compensatory tachycardia)
- 39. **Lightning bolt**: a large PE can cause cardiogenic shock (decreased cardiac output → hypotension)
- 40. **Black cat running down spiral staircase**: PE can be diagnosed with spiral CT angiography
- 41. **Heparin hunter**: anticoagulation (heparin, warfarin, etc.) is the treatment for DVT (and PE)
- 42. **Barricade buster**: thrombolytic agents (e.g. tissue plasminogen activator [tPA]) can be administered to dissolve the thrombus in severe PF
- 43. Grandfather clock surrounded by tense arterial net: chronic thromboembolic pulmonary hypertension can be a complication of recurrent PEs (leads to increased risk of RIGHT heart failure



- 1. **"METropolitan bus** : metastases are the most common cancers in the lung
- 2. Crab bra: breast cancer commonly metastasizes to the lung
- 3. Colon belt: colon cancer commonly metastasizes to the lung
- 4. Kidney purse: renal cancer commonly metastasizes to the lung
- Risky red dice: risk factors for developing lung cancer (e.g. smoking, radiation, pulmonary fibrosis, toxins)
- Smoking: most important risk factor for developing lung cancer (20x increased risk)
- 7. Radiation symbol: radiation to chest (lung cancer risk factor)
- 8. Fibrous pulmonary tree: pulmonary fibrosis (lung cancer risk factor)
- Toxic insulation: workplace exposures, i.e. asbestos, radon, metals, and aromatic hydrocarbons (lung cancer risk factors)
- 10. Gasping: lung cancer can present with dyspnea
- 11. Thin arm: lung cancer can present with weight loss
- 12. Falling food: lung cancer can present with decreased appetite
- 13. Clutching chest: lung cancer can present with chest pain (especially in younger patients)
- 14. Coughing warden: lung cancer can present with coughing (especially central tumors)
- 15. Wheezy party blower: lung cancer can present with wheezing (especially central tumors)
- 16. **Recurrent bacterial lanterns**: lung cancer can present with recurrent pneumonia (especially central tumors)
- 17. Bloody air duct : lung cancer can present with hemoptysis (especially central tumors)
- 18. Small prison cell: small cell lung carcinoma
- Squamous epithelial tile: squamous cell carcinoma (non-small cell lung carcinoma)
- 20. "Dining Den": adenocarcinoma (non-small cell lung carcinoma)
- 21. Large prison inmate: large cell carcinoma (non-small cell lung carcinoma)
- 22. "Sentral Cell Block": centrally located tumors include Small cell and Squamous cell carcinoma
- 23. "No smoking" in the Dining Den : adenocarcinoma is the most common type of lung cancer in non-smokers

- 24. **Young lunch lady**: adenocarcinoma is the most common lung cancer in women and patients under 40
- 25. **Smoker in the Dining Den**: adenocarcinoma is the most common type of lung cancer (in smokers and non-smokers)
- 26. **Glandular hair net**: adenocarcinoma will show glandular characteristics on histology (e.g. acinar, papillary, mucinous)
- 27. **Behind the glass plate**: adenocarcinoma in situ (AIS) has not yet crossed the basement membrane
- 28. Layer lining food containers: AIS consists of tall columnar cells spreading along alveolar septae (appears to thicken alveolar walls)
- 29. **Leopard print**: surface alveolar growth (as seen in AIS) is called a LEPIDIC growth pattern
- 30. Coughing up mucus : cells in AIS can be mucinous \rightarrow mucus production
- 31. **Mucus blob on chest**: AIS may present like pneumonia on CXR (hazy consolidation)
- 32. **Jello cubes beyond glass barrier**: adenocarcinoma has cuboidal to low columnar cells (hyperchromatic with prominent nuclei)
- 33. Coughing up mucus : adenocarcinoma cells can produce mucin → copious sputum production
- 34. **Columnar cells**: normal respiratory epithelium is pseudostratified and columnar
- 35. Temporary metal plates: columnar epithelium can be replaced with more resistant stratified squamous cells (reversible squamous metaplasia)
- 36. **Disgusting squamous tiles**: squamous metaplasia can progress to dysplasia (disordered squamous cells with hyperchromasia and mitotic figures)
- 37. **Cells breaking through floor**: squamous cell carcinoma in situ can progress to invasive carcinoma (invades basement membrane)
- 38. Pearl necklaces: well-differentiated squamous cell carcinoma exhibits keratin pearls and intercellular bridges
- 39. **Necrotic skull in cavity**: squamous carcinoma may exhibit central necrosis and cavitation



- 40. **Sheets of bubble wrap**: small cell carcinoma contains sheets of round blue (basophilic) cells with scant cytoplasm
- 41. **Granite cell**: small cell carcinoma has granular chromatin ("salt and pepper"), and stains positive for chromogranin (neuroendocrine marker)
- 42. **Neuroendocrine wiring**: small cell carcinoma is a neuroendocrine tumor
- 43. **Escaping prisoner**: small cell carcinoma metastasizes early (discovered diffusely in both lungs)
- 44. Radiation window: small cell carcinoma is sensitive to radiation treatment
- 45. Chemistry set: small cell is sensitive to chemotherapy
- 46. Large prisoner: large cell carcinoma contains large undifferentiated anaplastic cells (with large nuclei and prominent nucleoli)
- 47. **Wet pleural shirt and pericardial case**: lung cancer can cause pleural and pericardial effusions (regional tumor spread to pleura and pericardium)
- 48. **"Pancoast Airlines"**: Pancoast syndrome occurs with regional tumor spread to the superior pulmonary sulcus
- 49. Electric plexus fence : Pancoast tumors can invade the medial roots of the brachial plexus
- Shoulder pain: invasion of the brachial plexus causes shoulder pain, arm/neck pain, hand muscle wasting (pancoast tumor)
- 51. Air raid horn: Horner's syndrome (ptosis, miosis, anhidrosis) occurs with regional tumor spread to the sympathetic chain ganglia (pancoast tumor)
- Constricted horn : constricted pupil (miosis) seen in Horner's syndrome
- 53. Droopy search light: ptosis seen in Horner's syndrome
- 54. Mediastinal mast: lung cancer may extend medially and involve mediastinal structures
- 55. Horse with laryngeal reigns: hoarseness due to recurrent laryngeal nerve involvement (regional tumor spread to mediastinum)
- 56. **Red balloon face**: SVC syndrome → compression of the superior vena cava causes swelling of the face, neck, and upper extremities (regional tumor spread to mediastinum)

- Inappropriately wet head: SIADH is a paraneoplastic syndrome associated with small cell carcinoma (ADH release from tumor)
- 58. Antibody keys and empty calci-yum cups: lambert eaton myasthenic syndrome (LEMS) is a paraneoplastic syndrome associated with small cell carcinoma (antibodies against voltage-gated calcium channels)
- 59. Acetyl-cola trash bin : LEMS causes decreased acetylcholine release due to blocked presynaptic calcium channels → proximal muscle weakness
- 60. Struggling to get up: LEMS causes symmetrical proximal muscle weakness (decreased acetylcholine release)
- 61. Antibody keys under cerebral turban : neurological paraneoplastic syndromes associated with SCC (e.g. cerebellar degeneration, encephalomyelitis) are due to autoimmune response against antigens in neural tissue
- 62. **Cushion**: Cushing's syndrome is a paraneoplastic syndrome associated with small cell carcinoma (ACTH-like substance release from tumor)
- Clubbed fingers: hypertrophic osteoarthropathy (HOA) is a paraneoplastic syndrome associated with adenocarcinoma (digital clubbing, arthropathy)
- 64. **Wrapped joints**: HOA causes sudden arthropathy of the hands and wrists (less commonly elbows, knees, ankles)
- 65. Raised calcium cup : humoral hypercalcemia of malignancy is a paraneoplastic syndrome associated with sâ€□ca++â€□mous cell carcinoma (PTHrP release from tumor)
- 66. **Death graffiti**: though not the most common cancer, lung cancer is associated with the highest mortality rate
- Knocked out PhD: humoral hypercalcemia of malignancy is associated with suppressed PTH levels (PTH-independent hypercalcemia)
- 68. PhD disguise: humoral hypercalcemia of malignancy is caused by parathyroid hormone-related protein (PTHrP) release from tumor (PTHindependent hypercalcemia)

Renal Pathology 1.1 **Renal Function Tests & AKI**



- 1. Credit card: creatinine (used to measure kidney function)
- 2. CREATE your own MEAT combo: creatinine is the breakdown product of CREATINE (found in muscle cells)
- 3. "\$1 MENU": creatinine has a normal serum concentration of ~1.0 mg/dL (ranging from 0.6 to 1.2)
- 4. "Free filtered": creatinine is freely filtered at the glomerulus
- 5. "NEVER REABSORBED!" : once in the nephron, creatinine is NOT resorbed and only minorly secreted (Cr filtered load ≈ Cr urinary excretion)
- 6. "*May contain minor secretions": creatinine is secreted in small amounts into the nephron \rightarrow urine conc. is slightly higher than serum conc. (slightly overestimate GFR)
- 7. "Grounds Filter Rate": glomerular filtration rate (GFR) is the amount of fluid that filters across the glomerulus into Bowman's space (~125ml/min)
- 8. Credit card cleared: creatinine clearance can be used to approximate GFR (slight overestimation) because Cr is freely filtered at the glomerulus and is neither secreted (almost!) nor reabsorbed
- 9. "CUP \rightarrow " : C=UV/P (creatinine clearance equation used to estimate GFR)
- 10. "100% real": creatinine clearance (estimation of GFR) is normally >100 ml/min mg/dL (<100 ml/min is abnormal, <10 indicates renal
- 11. Elderly man knocking over filtered coffee: GFR decreases with age (renal dosing of drugs)
- 12. BUN bag: Blood Urea Nitrogen (BUN) is cleared by the kidney (final breakdown product of nitrogen containing compounds)
- 13. "N" order cycle: BUN comes from the urea cycle in the liver (amino acid metabolism)
- 14. Pyrimidine hexagon titles: BUN also comes pyrimidine metabolism (nitrogenous bases)
- 15. "Order 10": BUN has a normal serum concentration of ~10 mg/dL (ranging from 7-20)
- 16. Spilled filtered coffee: a decrease in GFR (renal injury or dysfunction) will cause an increase in BUN
- 17. "Pro Car Track" absorbing BUN grease: once in the nephron BUN is actively reabsorbed at the Proximal Convoluted Tubule
- 18. MEGAKING: Acute Kidney Injury (acute pre-renal, renal, or postrenal azotemia)
- 19. Trickle next to credit card: Acute Kidney Injury (AKI) manifests as increased serum creatinine and/or decreased urine output (oliguria)

- 20. Ketchup hemorrhage: hypovolemia or hemorrhage can cause PRE-RENAL AKI (kidney is hypoperfused)
- 21. Failing heart balloon: low output states (e.g. heart failure, shock) can cause PRE-RENAL AKI (kidney is hypoperfused)
- 22. BUN grease reabsorption: urea resorption in the proximal tubule is INCREASED during low perfusion states → INCREASED BUN/Cr ratio
- 23. Lifted BUN bag over credit card : increased BUN/Cr ratio (>15) in PRE-RENAL AKI
- 24. FENa fries dropping: decreased fractional excretion of sodium (FENa <1%) in PRE-RENAL AKI (sodium is conserved during low perfusion states)
- 25. "UP/UP N away!" : UNaPCr/PNaUCr (FENa equation used to assess ability to reabsorb sodium)
- 26. Pepper in the consOSMents: PRE-RENAL AKI is associated with a UOsm > 500 (concentrated urine to conserve volume)
- 27. Necrotic chocolate straw: acute tubular necrosis (ATN) is the most common cause of INTRINSIC RENAL AKI
- 28. Blue spotted kidney tray: acute interstitial nephritis (AIN) causes INTRINSIC RENAL AKI
- 29. Dripping glomerular curly straw: glomerulonephritis can cause INTRINSIC RENAL AKI
- 30. Falling BUN Bag over Credit card : BUN/Cr ratio <15 in INTRINSIC RENAL AKI (but both BUN and Cr are elevated)
- 31. Elevated FENa fries: FENa is >2% in INTRINSIC RENAL AKI (unable to reabsorb sodium)
- 32. Empty condOSMents bottle: INTRINSIC AKI is associated with a UOsm <350 (unable to conserve water)
- 33. Elevated BUN bag over Credit card : the BUN/Cre is >15 in POST-RENAL AKI (back-pressure enhances urea reabsorption)
- 34. Obstructive spit balls: bilateral nephrolithiasis can cause POST-RFNAI AKI
- 35. BPH Fire hat: benign prostatic hyperplasia (BPH) can cause POST-RENAL AKI
- 36. Cancer crab buckle: cancer of the ureter, bladder, or urethra can also cause POST-RENAL AKI
- 37. Fallen BUN bag and Credit card: the BUN/Cr ratio can be decreased in severe or prolonged POST-RENAL AKI
- 38. Toys behind cracked kidney glass: AKI can cause hyperkalemia, acidosis, hypertension, and uremia (see chronic kidney disease video





- Necrotic muddy drain pipe: acute tubular necrosis (ATN) is the most common form of acute kidney injury (AKI)
- 2. Ischemic zombie: ATN is caused by ischemia of tubule cells
- 3. **Empty kidney water gun**: ATN is usually caused by decreased renal perfusion (prerenal AKI)
- 4. **Bloody wound** : severe blood loss → systemic hypoperfusion → prerenal AKI →ischemic damage to kidney → ATN
- Broken heart string : MI → systemic hypoperfusion → prerenal AKI →ischemic damage to kidney → ATN
- Constricted red exhaust pipe: ischemia damages endothelial cells → decreased nitric oxide (vasodilator) and increased endothelin (vasoconstrictor) production
- 7. **Constricted red sleeve**: ischemic injury to the endothelium of the afferent arteriole leads to vasoconstriction
- 8. **Zombies stuck at AFFERENT entry way**: vasoconstriction of the damaged afferent endothelium impedes blood flow to the glomerulus
- 9. **Broken grounds filter rate** : glomerular filtration rate (GFR) is decreased in ATN
- Muddy epithelial leaves: ATN causes "muddy brown" casts in the urine sediment (sloughed tubular cells)
- 11. **Obstructing epithelial leaves**: sloughed epithelial cells accumulate in the tubular lumen, causing obstruction
- 12. Mud spilling on grounds filter rate : obstruction of tubular lumen by sloughed tubular cells in ATN further reduces GFR
- 13. **Broken down PRO CART TRACK**: The proximal convoluted tubule is particularly susceptible to ischemic injury in ATN
- 14. **Broken down loop-de-loop**: The thick ascending limb of the loop of henle is particularly susceptible to ischemic injury in ATN
- 15. **Patchy tube lining**: in ATN, the nephron will show dilated tubules with a patchy loss of epithelial cells
- 16. Broken tube edge and rusty holes: in ATN, sections of the nephron will show ruptured basement membrane and vacuolization of epithelial cells
- 17. "Come on IN": the initial insult (e.g. MI, sepsis, hemorrhage) occurs during the INITIATION phase of ATN (lasts ~ 36 hours)

- 18. **Smiling clown face**: the INITIATION phase of ATN is associated with fairly normal kidney function and urine output
- Maintenance shed: AKI develops during the MAINTENANCE phase of ATN with severe metabolic derangements and reduced urine output (lasts ~1-2 weeks after initial insult)
- 20. Cracked kidney on maintenance shed: the maintenance phase of ATN is associated with symptoms of AKI (e.g. oliguria, increased creatinine, fluid overload, metabolic abnormalities)
- 21. BUN bag: BUN is elevated during the maintenance phase of ATN
- 22. **Credit card slot**: creatinine is elevated during the maintenance phase of ATN
- 23. **Trickle**: the maintenance phase of ATN is associated with oliguria (<400mL/24hr)
- 24. Covering up with epithelial jacket: tubular re-epithelialization occurs during the recovery phase of ATN (!1-2 weeks after initial insult)
- 25. **Wet crotch**: the recovery phase of ATN is marked by profound diuresis (urine output up to 3-5 L/day)
- 26. Spilled banana peels, peanut shells, Calci-Yum cups, and magazine trash: the recovery phase of ATN can cause electrolyte abnormalities (e.g. hypokalemia, hyponatremia, hypocalcemia, and hypomagnesemia)
- 27. **Toxic waste at the Pro Cart Track**: the proximal convoluted tubule is the primary site of injury in NEPHROTOXIC ATN
- 28. Sai weapon: aminoglycosides can cause nephrotoxic ATN
- 29. **Chomped chicken leg**: damaged muscle (e.g. crush injury, rhabdomyolysis) can cause nephrotoxic ATN
- 30. Heme ninja stars : damaged muscle releases nephrotoxic heme pigments into the bloodstream → nephrotoxic ATN
- 31. Yin-yang: IV contrast can cause nephrotoxic ATN

Renal Pathology 1.3 Tubulointerstitial Nephritis



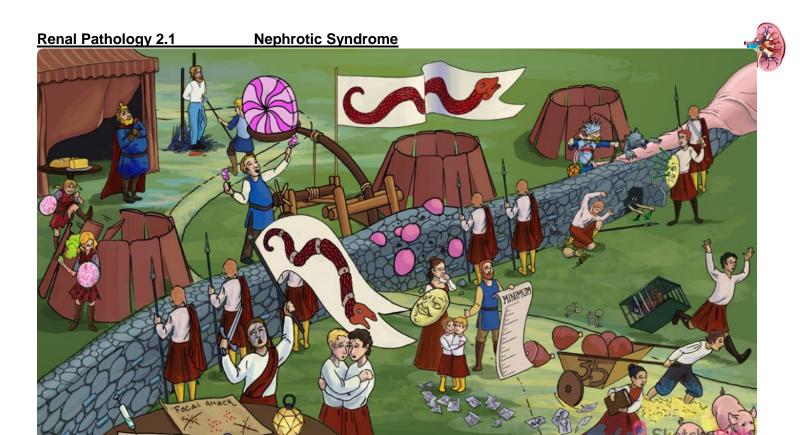
- Flaming kidney: tubulointerstitial nephritis (e.g. acute interstitial nephritis - AIN) is associated with acute renal interstitial inflammation
- 2. Anti-inflammatory fire extinguisher: NSAIDs can precipitate acute interstitial nephritis (AIN)
- 3. Furious kid under the loop de loop : furosemide (a loop diuretic) can precipitate AIN
- 4. Pencils in kidney: penicillin can precipitate AIN
- Stinky sulfur eggs: sulfonamide drugs (e.g. TMP/SMX) can precipitate AIN
- Degranulating bee hive: drug-induced interstitial nephritis can be mediated by a type I hypersensitivity reaction (cross-linking Ig-E on mast cells → release of proinflammatory substances)
- Helper squire running by cage: drug-induced interstitial nephritis can be mediated by a type IV ("delayed-type") hypersensitivity reaction (antigen presenting cells activate TH2 helper T-cells)
- 8. **Blue lights dotting kidney**: AIN shows interstitial edema with a diffuse inflammatory infiltrate on histology
- 9. Slingshot with pink granules: AIN can cause eosinophilia
- 10. **Eo-slingshot granules in puddle**: AIN can cause eosinophiluria
- 11. White knights, squires, and archers in puddle: AIN can present with white blood cell casts
- 12. **BUN bag and credit card**: AIN can present with elevated serum BUN and creatinine)
- 13. Trickling water: AIN can present with oliguria
- 14. Cracked kidney mirror: AIN causes intrinsic AKI (elevated serum BUN and creatinine and numerous metabolic and hemodynamic derangements)
- 15. Flaming head: AIN can present with fever
- 16. Spotted clown outfit: AIN can present with rash
- 17. **Delayed demolition**: AIN symptoms occur 1-2 weeks after inciting agent (type IV hypersensitivity reaction)
- 18. **Chronic grandfather clock** : chronic tubulointerstitial nephritis (TIN) (e.g. analgesic nephropathy)

- 19. **Anti-inflammatory fire extinguisher**: chronic NSAID use can cause analgesic nephropathy (chronic TIN)
- Kicking in the BACK: Chronic pain patients (e.g. BACK pain, migraines) are at risk for chronic TIN due to chronic NSAID use
- 21. **Pointy pyramids in renal tunnel**: NSAIDs accumulate at the renal papillae (the tips of medullary pyramids)
- 22. **Oxidizing sparks**: NSAIDs cause injury to the renal interstitium via free radical damage
- 23. Patchy blue sparks : \rightarrow NSAIDs cause patchy inflammation in the renal interstitium
- 24. Calcium deposits: NSAIDs cause calcification in renal papillae (in areas of chronic inflammation)
- 25. **Fibrotic bush**: chronic inflammation in analgesic nephropathy causes interstitial fibrosis
- 26. **Decaying pyramids in renal tunnel**: in analgesic nephropathy causes microvascular damage AND vasoconstriction of afferent renal vessels→ ischemia →renal papillary necrosis
- 27. Shrunken kidney pendulums : chronic TIN causes chronic kidney disease → bilaterally small scarred kidneys
- 28. **Lead paint cans**: chronic lead exposure can cause chronic TIN (i.e. lead nephropathy)
- 29. "Liftium": chronic lithium use can cause chronic TIN
- 30. **Antibody lights**: autoimmune diseases (e.g. SjĶgren syndrome, lupus) can cause chronic TIN



- 1. Endothelial shields: glomerular capillary (ENDOTHELIUM)
- 2. **The wall**: the glomerular basement membrane is thick, electron dense, and made of collagen, laminins, and glycoproteins
- 3. Minus signs: the GBM is normally negatively charged
- 4. **Foot soldiers**: podocytes (EPITHELIUM) have foot processes with filtration slits (selectively permeable)
- 5. **Supportive field**: the mesangium surrounds the capillaries and provides structural support to the glomerulus
- 6. Excretory river : Bowman's space \rightarrow renal tubule
- Falling minus bricks : injury to the basement membrane can cause loss of negative charge → NEPHROTIC syndrome
- Coins: in NEPHROTIC syndrome, injury to the glomerulus via cytokines (NOT cellular infiltrate, inflammation) →NEPHROTIC syndrome
- 9. **Meat cart #35**: NEPHROTIC syndrome is characterized by marked PROTEINURIA (> 3.5 grams/day)
- Falling album: NEPHROTIC syndrome is characterized by HYPOALBUMINEMIA due to loss of albumin into the urine (hyperalbuminuria
- Edematous king : hypoalbuminemia → decreased oncotic pressure
 → fluid shifts into interstitium → generalized edema (NEPHROTIC syndrome)
- Butter in front of liver tent: hypoalbuminemia → liver synthesis of proteins (including lipoproteins) → hyperlipidemia (NEPHROTIC SYNDROME)
- 13. Fat oval pigs: fatty casts and "oval fat bodies" in urine (lipiduria)
- 14. Dropping throm-beaver cage : proteinuria includes loss of antithrombin III \rightarrow hypercoagulable state
- 15. **Stained with red paint**: amyloid appears pink when stained with Congo red
- 16. Firewood thrombus : decreased antithrombin III → increased thrombin activity (hypercoagulable state)

- Stabbing in flank: hypercoagulable state → renal vein thrombosis (flank pain, gross hematuria)
- 18. Wormy left pant leg: left renal vein thrombosis can cause left varicocele
- 19. Falling antibody keys : proteinuria includes loss of gamma-globulins in the urine → hypogammaglobulinemia → increased risk of encapsulated bacterial infections
- 20. Frothy river: massive proteinuria can cause frothy urine
- 21. "MINIMUM": minimal change disease primarily affects children and causes "minimal" changes on histology (normal light microscopy, no immune complexes on immunofluorescence)
- 22. **Hugging kid foot soldiers**: minimal change disease causes podocyte effacement and fusion (visible on electron microscopy)
- 23. Cowering footsoldier : podocytes effacement, slit diaphragm disruption, depletion → NEPHROTIC SYNDROME
- 24. Blowing nose: minimal change disease develops several weeks after an upper respiratory infection, allergic reaction, insect sting, or immunization
- 25. Photos falling from album: minimal change disease causes selective leakage of albumin ("selective albuminuria") due to loss of negative charge on the basement membrane
- 26. Moon face : minimal change disease responds glucocorticoids



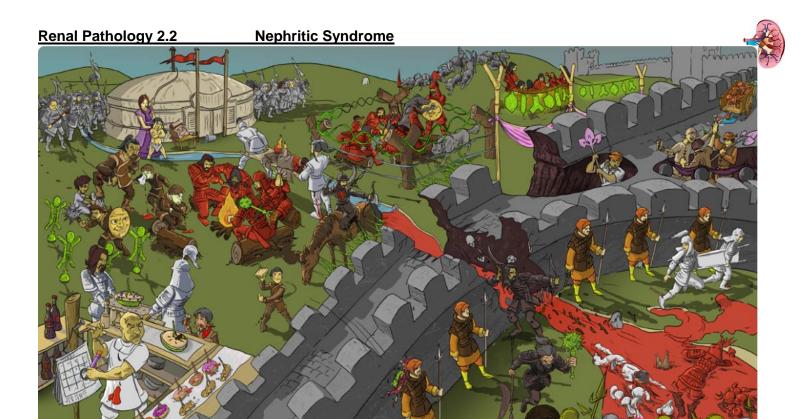
- 27. **"FOCAL ATTACK"**: focal segmental glomerulosclerosis (FSGS) is "focal" (affecting only some glomeruli)
- 28. "SEGMENTAL ATTACK": focal segmental glomerulosclerosis (FSGS) is "segmental" (affecting only some segments of a glomerulus)
- 29. **SCALY sclerotic snake**: focal segmental glomerulosclerosis (FSGS) causes sclerosis (light microscopy shows: obliterated capillaries with hyalin deposition)
- 30. **Hugging foot soldiers**: FSGS causes podocyte effacement and fusion (visible on electron microscopy)
- 31. Cracked kidney rocks: FSGS frequently causes end stage kidney disease
- 32. **Virus lantern**: FSGS can be caused by viral infections (HIV, hepatitis)
- 33. **Syringe**: FSGS can be caused by heroin abuse or systemic disease (diabetes, hypertension, vasculitis, sickle cell)
- 34. **Glycosylated kidney lollipops**: diabetic nephropathy generally causes a nephrotic syndrome
- 35. Wall thickened by pink deposits: early on, diabetic nephropathy causes basement membrane thickening (due to nonenzymatic glycosylation)
- 36. **Big pink lollipop in mesangial field**: later stage of diabetic nephropathy causes nodular glomerulosclerosis, characterized by Kimmelstiel-Wilson nodules (light microscopy shows pink round deposits of laminated mesangial matrix)
- 37. **SCALY sclerotic snake**: nodular glomerulosclerosis (diabetic nephropathy) causes sclerosis (light microscopy shows: obliterated capillaries with hyalin deposition)
- 38. **Wall thickened by long cape**: membranous glomerulopathy causes diffuse thickening of the glomerular basement membrane (visible on light microscopy)
- 39. **Antibody arrows injuring foot soldier**: membranous nephropathy can be caused by autoantibodies against the phospholipase A2 receptor on podocytes →leads to subepithelial antibody deposits)

- Dome-like helmet decorated with spikes: subepithelial deposits have a "spike and dome" appearance on electron microscopy (in membranous glomerulopathy)
- 41. Wall engulfing antibody arrows: "spike" appearance is due to the basement membrane engulfing "domes" of subepithelial immune deposits (visible on electron microscopy in membranous glomerulopathy)
- 42. **Crab chest plate**: membranous nephropathy can be secondary to solid tumors (lung, breast, prostate, colon)
- 43. **Viral quiver**: membranous nephropathy can be secondary to viral infections (hepatitis B or C)
- 44. **Lupus wolf**: membranous nephropathy can be secondary to lupus erythematosus
- 45. **Fluorescent green arrows**: immunofluorescence microscopy shows granular deposits of immunoglobulin and complement along the basement membrane (in membranous glomerulopathy)
- 46. **Green perfume bottle**: amyloid displays apple green birefringence in polarized light
- 47. Moon shield: membranous nephropathy can be treated with corticosteroids
- 48. Armored lady: amyloidosis (a systemic disorder that involves deposition of misfolded proteins in tissues) can cause nephrotic syndrome
- 49. Armoured lady in mesangial field: in early renal amyloidosis, there are focal amyloid deposits within the mesangium
- Armoured lady in capillary circle: in later renal amyloidosis, amyloid obliterates glomerular capillaries



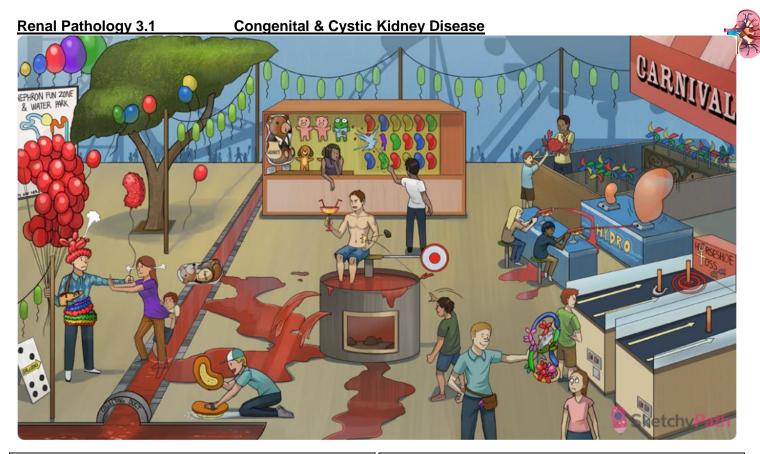
- Supportive field: the mesangium surrounds the capillaries and provides structural support to the glomerulus
- 2. Circle of endothelial warriors: glomerular capillary (ENDOTHELIUM)
- 3. The Great Wall: the glomerular basement membrane
- 4. **Footsoldiers**: podocytes (EPITHELIUM) have foot processes with filtration slits (selectively permeable)
- 5. Excretory river : Bowman's space \rightarrow renal tubule
- Endothelial warriors around fire: in nephritic syndrome, glomerular injury is a result of inflammation
- 7. First responders carrying away endothelial warrior : inflammatory infiltrate (including neutrophils) → glomerular capillary damage → hematuria and AKI (nephritic syndrome)
- 8. **Blood in river** : nephritic syndrome is characterized by hematuria (gross or microscopic)
- Dysmorphic red soldier: nephritic syndrome presents with dysmorphic RBCs (hallmark of glomerular injury)
- 10. **Collecting red helmets**: nephritic syndrome presents with RBC casts (hallmark of glomerular injury)
- 11. White soldier in river: nephritic syndromes can present with WBC in the urine sediment
- Collecting white helmets: nephritic syndrome can present with WBC casts
- 13. Trickle: nephritic syndromes can present with oliguria (AKI)
- 14. **High pressure steam**: nephritic syndromes can present with hypertension (due to salt and volume retention)
- 15. **Puffy face**: nephritic syndromes can cause periorbital (and less commonly peripheral) edema (due to salt and volume retention)
- 16. **Raised BUN bag**: nephritic syndrome can present with an elevated BUN and creatinine (AKI)
- 17. **Dropped meats**: nephritic syndrome causes proteinuria (>150 mg/day less than nephrotic range of 3.5 g/day)
- 18. IgA dummy soldiers in the field : IgA nephropathy (Berger disease) is caused by deposition of IgA and IgA immune complexes in the mesangium)
- Blowing nose and grabbing stomach: IgA nephropathy may present 1-2 days after an upper respiratory or GI infection (abnormal IgA synthesis and glycosylation)

- 20. Berger: Berger disease (IgA nephropathy)
- 21. **Blood trickling**: IgA nephropathy usually presents with gross hematuria that lasts for several days
- 22. **Periodic blood puddles**: patients with IgA nephropathy may have episodic hematuria
- 23. **Shoeshine**: Henoch-Schonlein purpura (HSP) causes a renal disease similar to IgA nephropathy
- 24. Proliferating army in the field: focal or diffuse mesangial proliferation (IgA nephropathy, post-strep, diffuse proliferative, membranoproliferative glomerulonephritis, dense deposit disease
- 25. **Granular green glow**: immunofluorescence shows a granular pattern in the mesangium due to IgA immune complex deposition
- 26. **Moon-face shield**: IgA nephropathy can be treated with glucocorticoids
- 27. **Pyogenes pie** : post-streptococcal glomerulonephritis (PSGN)
- Membranous sash along wall: membranoproliferative glomerulonephritis (MPGN) causes diffuse GBM thickening
- 29. **Pie on face and neckerchief**: PSGN can occur after group A strep (Strep. pyogenes) infection, including pharyngitis OR skin infection
- 30. **Pie in mesangial field**: PSGN is associated with immune complex deposition in the mesangium
- 31. **Pie behind endothelial soldiers**: PSGN is associated with subendothelial immune complex deposition
- 32. Pie landing on the back of epithelial foot soldier: PSGN is associated with subepithelial immune complex deposition
- 33. "Week 3" on calendar: post-streptococcal glomerulonephritis (PSGN) occurs 1-3 weeks after a skin or pharyngeal infection with nephritogenic strains of group A strep
- 34. Cola bottles: hematuria in PSGN is often described as "cola-colored"
- 35. First responders with blueberry pie: in PSGN, light microscopy shows marked leukocyte infiltration (lots of nuclei present) in the mesangium and endothelium
- 36. **Granular green pie tins**: in PSGN, immunofluorescence microscopy shows granular pattern (due to IC deposition)
- 37. Lysed donuts and helical donuts: serum antistreptolysin-O (ASO) and anti-DNase B titers may be elevated after a group A strep infection (ASO less likely with skin infection)



- 38. **Diffusely proliferating lupus wolves** : diffuse proliferative glomerulonephritis is the most common presentation of lupus nephritis
- 39. **Antibody posts holding double helix**: DNA anti-DNA immune complexes (seen in diffuse proliferative glomerulonephritis)
- Double helical fence around endothelial soldiers: DNA anti-DNA immune complexes deposit in the subendothelial space (diffuse proliferative glomerulonephropathy)
- 41. Looped wire around endothelial soldiers: light microscopy shows "wire looping" of the capillaries due to subendothelial immune complex deposition (diffuse proliferative glomerulonephritis)
- 42. **Green granular glow**: immunofluorescence shows a granular pattern due to immune complex deposition
- 43. **Moon-face shield**: treatment of diffuse proliferative glomerulonephritis with glucocorticoids and cyclophosphamide may slow progression to chronic kidney disease
- 44. **Lobulated shovel**: light microscopy shows hypercellularity and enlarged, lobular glomeruli (MPGN)
- 45. **Wall splitting**: electron and light microscopy show splitting of the glomerular basement membrane due to ingrowth of mesangium ("tram tracking") (MPGN)
- 46. **Viral, bacterial, and antibody lanterns**: MPGN can be caused by viral infections (hepatitis B or C), bacterial infections (endocarditis, shunt nephritis), or autoimmune diseases (due to chronic IC formation)
- 47. IC lanterns behind endothelial soldiers: immune complexes deposit in subendothelial space (MPGN)
- 48. IC lanterns behind endothelial soldiers: immune complexes deposit in subendothelial space (MPGN)
- 49. **Dense bomb deposits**: dense deposit disease involves deposition of a material of unknown composition in the basement membrane
- 50. **Dense ribbon**: the GBM eventually transforms into a long irregular and extremely electron-dense ribbon (DDD)
- 51. Excessive complements: dense deposit disease is associated with overactivation of the alternative complement pathway
- 52. **3 friendship bracelets**: dense deposit disease is associated with the formation of the autoantibody, C3 nephritic factor → stabilized C3 convertase → overactive alternative pathway (low C3 with normal C4)

- Deadly crescent weapon: rapidly progressive glomerulonephritis (RPGN) causes crescents in the glomerulus (rapid decline in kidney function)
- 54. Deadly IC mace: immune-complex mediated glomerulonephritides (e.g. PSGN, diffuse proliferative glomerulonephritis, IgA nephropathy, HSP) can lead to RPGN
- 55. Decaying break in wall : on light microscopy, RPGN glomeruli will show segmental necrosis and breaks in the glomerular basement membrane
- 56. **Scattered fibrous twigs**: plasma proteins and fibrin deposit in the crescents formed in RPGN
- 57. Advancing epithelial army: crescent formation in RPGN is caused by epithelial cell proliferation from the capsule (parietal) → obliteration of Bowman's space
- 58. **Green granular glow**: immunofluorescence microscopy shows a granular pattern in the immune complex glomerulonephritides progressing to RPGN
- 59. Crescent warrior shooting ankle: pauci-immune RPGN (no IC deposition) may be associated with anti-neutrophilic autoimmune vasculitides like Wegener's granulomatosis (c-ANCA) and microscopic polyangiitis (p-ANCA)
- 60. Crescent warrior shooting wall: anti-GBM antibody-mediated RPGN (e.g. Goodpasture's disease) is caused by antibodies directed against antigens in the GBM
- 61. Linear layer of green arrows: anti-GBM antibody-mediated RPGN shows a linear pattern on immunofluorescence microscopy
- 62. Blood on chest: while anti-GBM can be isolated to the kidney, involvement of the pulmonary capillary bed leads hemoptysis (Goodpasture's syndrome)
- 63. "IV": anti-GBM antibody-mediated RPGN involves IgG autoantibodies directed against type IV collagen in the GBM (also pulmonary capillaries in Goodpasture's)



- Horseshoe game: horseshoe kidney (the most common congenital renal malformation)
- 2. **Caught on inferior post**: during embryologic ascent, a horseshoe kidney gets stuck at the inferior mesenteric artery
- 3. **X shaped pinwheel toys**: Turner syndrome (monosomy X) is associated with horseshoe kidney
- 4. Cancer crab toy: horseshoe kidney increases risk of Wilms tumor
- 5. **Stones in the dunk tank**: congenital and cystic disorders of the kidney (e.g polycystic kidney disease) increased risk of kidney stones
- Red dunk tank water: congenital and cystic disorders of the kidney (e.g polycystic kidney disease) can present with hematuria and flank pain (due to kidney stones)
- Infected bladder cup: congenital and cystic disorders of the kidney (e.g polycystic kidney disease) can increase risk of urinary tract infections
- 8. Recurring bacterial lanterns: congenital and cystic disorders of the kidney (e.g polycystic kidney disease) can cause recurrent urinary tract infections
- 9. **Dysplastic bunch of balloons**: renal dysplasia (abnormal development of one or both kidneys) is the most common cystic disease in children
- 10. **Dysplastic shark toy**: renal dysplasia is associated with abnormal mesenchymal tissue (e.g. cartilage and bone)
- 11. **Popped kidney balloon**: renal agenesis (congenital absence of renal parenchyma)
- 12. **Tightly wrapped Potter the bear**: bilateral renal agenesis can result in oligohydramnios → Potter sequence (flattened nose, clubfeet, lung hypoplasia)
- 13. Kid hiding from bunch of kidney balloons: autosomal recessive polycystic kidney disease (ARPKD - symptomatic in early childhood)
- 14. Balloon with little cysts: ARPKD can present with bilaterally enlarged kidneys with diffuse small cysts on fetal ultrasound
- 15. **Corduroy fibers**: ARPKD is caused by a mutation in PKHD1 (fibrocystin gene)
- 16. Stepping in collecting duct: in ARPKD, cysts develop in the collecting ducts

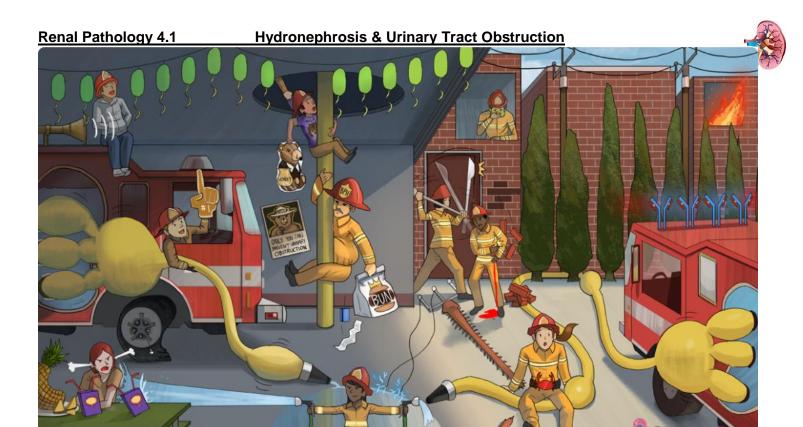
- 17. Square tiles lining collecting duct: in ARPKD, cysts are lined by cuboidal epithelium
- 18. Tightly wrapped Potter the bear : ARPKD can cause oligohydramnios → Potter sequence (flattened nose, clubfeet, lung hypoplasia)
- 19. Bulging flank: ARPKD can present with bilateral flank masses
- 20. **High pressure steam**: ARPKD can cause hypertension in first months of life
- 21. Balloons caught in liver tree: ARPKD can cause cystic dilation of intrahepatic bile ducts, hepatomegaly, and hepatic fibrosis
- 22. **Domino**: autosomal dominant polycystic kidney disease (ADPKD usually presents in adulthood)
- 23. **Polygon pattern**: ADPKD is caused by a mutation in PKD1 or PKD2 (less common) which code for polycystin-1 and polycystin-2
- 24. **Nephron fun zone sign**: cysts in ARPKD develop throughout the nephron
- 25. Growing balloons: cysts in ARPKD grow larger over time
- 26. **High pressure steam**: ADPKD can cause hypertension
- 27. **Popping balloon hat** : ADPKD can cause berry aneurysms in the circle of willis → subarachnoid hemorrhage
- 28. **Balloons caught in liver tree** : ADPKD can also present with hepatic cysts
- 29. Pancreatic balloon bag : ADPKD can also present with pancreatic cysts
- 30. Balloon belt: ADPKD can also present with diverticulosis
- 31. **Central sponge gloves** : medullary sponge kidney (multiple cysts in renal medulla)
- 32. Cleaning the end of the collecting duct: the cysts in medullary sponge kidney develop along the terminal collecting duct
- 33. **Dilated kidney water balloon**: horseshoe kidney can cause hydronephrosis

Renal Pathology 3.2 Chronic Kidney Disease (CKD)



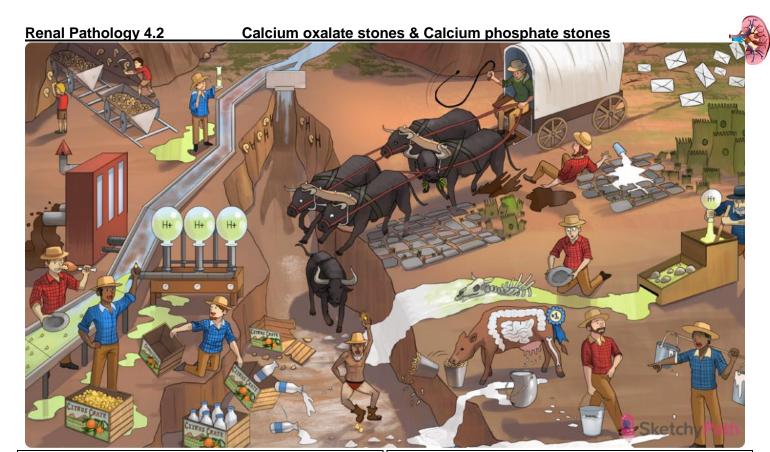
- Fossilized kidney-shaped eggs: chronic kidney disease (CKD) (kidneys appear bilaterally shrunken with a red-brown, diffusely granular surface)
- 2. Empty grounds filter rate: CKD presents with decreased GFR
- 3. **Coffee levels 1-5**: the 5 stages of CKD are defined by GFR, with stage 5 being end-stage renal disease requiring dialysis
- Credit card : CKD presents with increased creatinine (decreased GFR
 → decreased Cr clearance)
- 5. Photo album: CKD presents with albuminuria due to kidney damage
- 3 month expedition: CKD is diagnosed by 3 months of reduced GFR or elevated urine albumin
- DiaSweeties candies: poorly controlled diabetes one of the MOST COMMON causes of CKD (due to microvascular damage)
- 8. **High pressure steam**: chronic hypertension is one of the MOST COMMON causes of CKD (due to microvascular damage)
- 9. Frayed glomerular knots: chronic glomerulonephritis can cause CKD
- Kidney shaped bunch of balloons: polycystic kidney disease can cause CKD
- Constricting red kidney straps: bilateral renal artery stenosis can cause CKD (due to ischemic nephropathy)
- 12. **Pineapples**: chronic pyelonephritis can cause CKD (due to progressive renal scarring)
- 13. Chomping glomerular reeds: initial injury leading to CKD
- 14. **Tall glomerular reeds in the river**: the remaining healthy glomeruli increase filtration to preserve GFR (adaptive hyperfiltration)
- 15. **Dead glomerular reeds**: the remaining hyperfunctioning glomeruli eventually become damaged by the extra load (labs start to show CKD)
- 16. **Dry sclerotic glomerular reeds**: CKD shows advanced scarring of the glomeruli on histology
- 17. Fibrotic kidney-shaped nest: CKD eventually leads to interstitial fibrosis
- Acid volcano: CKD can cause metabolic acidosis due to impaired hydrogen excretion, impaired bicarbonate reabsorption, and accumulation of uric acid
- 19. **Elevated bananas**: CKD can cause hyperkalemia (due to decreased filtration of potassium due to decreased filtration of potassium and the H+/K+ buffering system with metabolic acidosis)

- 20. **Wet body**: CKD can cause fluid retention (leading to both diffuse and pulmonary edema)
- 21. Wax arm in water: CKD can present with waxy casts in the urine
- 22. **High pressure steam**: CKD can cause hypertension (due to volume overload)
- 23. Cracked draining kidney: CKD can cause a normocytic anemia (due to decreased erythropoietin production)
- 24. "P" fossils: CKD can cause hyperphosphatemia (due to decreased filtration and increased bone resorption in CKD-bone mineral disease)
- 25. Falling calci-yum ice cream: CKD can cause hypocalcemia (due to decreased vitamin D production and hyperphosphatemia)
- 26. Falling "D― : CKD can cause hypovitaminosis D (due decreased activity of 1-alpha-hydroxylase, and decreased production of 1,25-dihydroxyvitamin D)
- 27. **PthD paleontologist**: CKD can cause secondary hyperparathyroidism (hypocalcemia stimulates the parathyroid gland to release parathyroid hormone (PTH))
- 28. Bone fossil with dirt-filled holes: secondary hyperparathyroidism causes CKD-mineral and bone disorder (CKD-MBD) → osteitis fibrosa cystica (bone resorption causes cystic "brown tumors" that fill with fibrosis and hemosiderin)
- 29. **Crumbling skeleton from Malaysia**: secondary hyperparathyroidism and decreased vitamin D causes CKD-MBD → osteomalacia (decreased mineralization of bone osteoid)
- 30. "JUREASSIC": CKD can cause uremia (elevated BUN)
- 31. Vomiting: uremia can cause nausea and vomiting
- 32. Flapping wings: uremia can cause asterixis (tremulousness) and other serious neurologic effects
- 33. **Broken plates** : uremia can cause platelet dysfunction → pathologic hemorrhage throughout the body
- 34. Cracked heart shell: uremia can cause serous pericarditis (or hemorrhagic if comorbid with platelet dysfunction)
- 35. **Brain-head dino**: uremia can cause significant neurologic symptoms (e.g. peripheral neuropathy, encephalopathy, seizure, coma, death)
- 36. Little itchy mammal: uremia can cause severe pruritis
- Clogged coronary crown: CKD is an independent risk factor for developing coronary artery disease



- Dilated pelvic and calyceal hoses: hydronephrosis (dilated renal pelvis and calyces)
- 2. **Crushed deflated tire**: hydronephrosis can cause pressure atrophy of the renal medulla and cortex
- 3. **Tarnished papillary hubcap** : sudden onset hydronephrosis can cause renal papillary necrosis
- 4. Horn sound: hydronephrosis can be diagnosed with ultrasound
- Abdominal pocket mass: newborns with hydronephrosis can present with an abdominal mass
- Kid kinking proximal hose: ureteropelvic junction obstruction is the most common cause of unilateral fetal hydronephrosis
- 7. **Puddle girl spraying back water**: vesicoureteral reflux (VUR) can cause hydronephrosis in children (unilateral or bilateral)
- 8. **Right angled spray nozzle**: perpendicular insertion of the ureters into the bladder can predispose to vesicoureteral reflux
- Bladder juice boxes: VUR predisposes to recurrent urinary tract infections (UTIs)
- Renal pineapple : VUR predisposes to chronic pyelonephritis → scarring
- 11. **High pressure steam**: VUR can cause hypertension (scarring from chronic pyelonephritis leads to renal insufficiency)
- 12. **Obstructing top of urethral pole**: posterior urethral valve can cause urinary tract obstruction in boys → hydronephrosis (bilateral)
- 13. Oh by all means, eat your fast food, enjoy your foam finger! Hey, even hack at a wall with a scalpel, guys...don't worry about this over here:
- 14. Manly wolf shirt: posterior urethral valve is made up of a wolffian duct remnant
- 15. Tightly wrapped Potter the bear : posterior urethral valve can cause oligohydramnios → Potter sequence (flattened nose, clubfeet, lung hypoplasia)
- 16. **Second dilated fire truck** : obstruction distal to the ureters can cause bilateral hydronephrosis (e.g. VUR, posterior urethral valve, BPH)
- 17. **Falling stones obstructing hose**: urolithiasis is the most common cause of urinary tract obstruction in adults (can cause unilateral hydronephrosis)

- 18. Slashing scalpel axe: the ureters can become damaged or unintentionally ligated after pelvic surgery (causing urinary tract obstruction and unilateral hydronephrosis)
- 19. **Recurrent bacterial lanterns**: urinary tract outflow obstruction can cause recurrent UTIs
- 20. Stone striking flank: acute urinary tract obstruction (e.g. urolithiasis, surgical injury) can present as sharp flank pain radiating to the ipsilateral groin
- 21. **Bleeding down pant leg**: acute urinary tract obstruction (e.g. urolithiasis, surgical injury) can present with gross hematuria
- 22. **Nauseated firefighter**: acute urinary tract obstruction (e.g. urolithiasis, surgical injury) can present with nausea and vomiting
- 23. **Crab buckle**: pelvic and abdominal cancers (e.g. bladder cancer, uterine cancer) can cause chronic urinary tract obstruction and hydronephrosis (unilateral or bilateral)
- 24. **Fibrous back wall of trees**: retroperitoneal fibrosis can cause chronic urinary tract obstruction and hydronephrosis (unilateral or bilateral)
- 25. 4 IgG lights: retroperitoneal fibrosis is associated with IgG-4 related systemic disease (including autoimmune pancreatitis, Riedel's thyroiditis, sclerosing aortitis)
- 26. Enlarged fire chief grabbing urethral pole: benign prostatic hyperplasia (BPH) is a common cause urinary tract obstruction in older men (can cause bilateral hydronephrosis)
- 27. **Broken spinal power lines**: neurogenic bladder (e.g. due to spinal cord injury, diabetes) can cause bilateral hydronephrosis
- 28. Fast food BUN bag and credit card : BILATERAL urinary tract obstruction can cause elevated serum BUN and creatinine (postrenal azotemia)



- Dried up river deposits: all renal stones form due to supersaturation (adequate hydration is cornerstone of treatment)
- Milk dripping into river bed : high concentration of calcium in the renal tubule → calcium stones (calcium oxalate most common)
- Ox in river bed : too much oxalate in the renal tubule → calcium oxalate stones
- 4. **Milk dripping from udders**: hypercalciuria (e.g. idiopathic, or due to hypercalcemia, chronic acidosis) can cause calcium stones
- 5. Normal milk bucket : hypercalciuria with normocalcemia \rightarrow calcium stones
- 6. **First place dairy cow**: hypercalciuria with normocalcemia is the most common metabolic abnormality causing calcium stones
- GI cow spot: absorptive hypercalciuria (excessive gut absorption of calcium) is the most common cause of hypercalciuria with normocalcemia
- 8. Leaking kidney milk bucket: renal hypercalciuria (defect in proximal reabsorption of calcium) can cause hypercalciuria with normocalcemia
- Bone cow spot : resorptive hypercalciuria (excess resorption of calcium from the bone) can cause hypercalciuria with normocalcemia
- Elevated milk buckets: hypercalcemia (e.g. due to PHPT, cancer) can cause hypercalciuria and calcium stones
- 11. **Acid on bones**: acidosis can cause hypercalciuria and calcium stones (calcium-phosphate buffer system)
- 12. Citrus crate with milk: citrate binds calcium in the renal tubules (soluble complex prevents stone formation
- Acid miners: acidosis can cause calcium stones (due to hypocitraturia)
- 14. Acid miner grabbing citrus crate : acidemia promotes resorption of citrate from tubule → less citrate bound to calcium in tubule → calcium stones
- 15. Spewing mud: diarrhea can cause calcium stones (due to volume depletion, and acidemia with hypocitraturia)
- Meat eater: high protein diets can cause calcium stones (acidemia causes hypocitraturia)

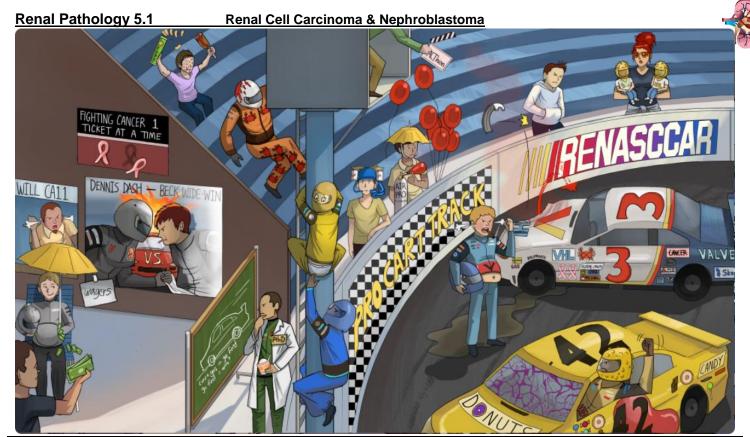
- 17. **Crushed citrus crate**: vitamin C deficiency can cause calcium stones (due to hypocirtaturia)
- 18. Cow eating salty peanuts: increased dietary sodium intake can cause calcium stones (reduced Na+ and Ca2+ reabsorption in the nephron through their symporter)
- Spilling milk: decreased dietary calcium promotes calcium oxalate stone formation (due to increased GI absorption of unbound oxalate)
- Oxen stampede: increased oxalate GI absorption (e.g. due to decreased dietary Ca2+, vegan diet, malabsorption) promotes calcium oxalate stone formation
- 21. Ox eating plants: pure vegan diets (without calcium supplementation) can cause calcium oxalate stones
- 22. Milk collecting in damaged GI path : fat malabsorption (e.g. Crohn's, short gut) binds calcium in the gut → GI absorption of unbound oxalate → calcium oxalate stones
- 23. Fossil mining kids: calcium phosphate stones are the most common stone found in children
- 24. **Acid cylinder**: type 1 renal tubular acidosis (RTA) promotes calcium phosphate stone formation (acidemia, plus alkaline urine environment)
- 25. **Elevated "pH" shape**: alkaline urine environment promotes calcium pHosphate stone formation
- 26. **Envelopes from ox cart** : calcium oxalate crystals have an "envelope" shape on microscopy
- 27. **Wedge shaped fossil mine car**: calcium phosphate crystals have an "elongated wedge" shape on microscopy
- 28. **Pale thighs**: hydrochlorothiazide can help prevent calcium stone formation by increasing reabsorption of calcium in the distal tubule

Renal Pathology 4.3 Magnesium Ammonium, Phosphate (MAP) Stones, Uric Acid Stones & Cystine Stones



- Frontier MAP: magnesium-ammonium-phosphate (MAP) renal stones (second most common in adult)
- 2. Stag antlers: MAP stones can present as struvite or "staghorn" calculi
- 3. **Urease spray**: MAP stones can form during UTI with urease positive organism (urea → ammonium + CO2)
- Blue puddles: basic urine decreases the solubility of phosphate → MAP stone formation
- Goblet with ureteral straws: MAP stones can form during a UTI with a urease positive organism (e.g. Proteus, S. Saprophyticus, Klebsiella)
- Coffin lid: MAP crystals have a characteristic "coffin lid" or rectangular prism shape on microscopy
- 7. Needles in uric acid yarn : uric acid (urate) stones
- 8. **Yarn ball on toe**: gout causes hyperuricemia → uric acid stones
- Raised white archers and knights: conditions with rapid cell turnover (e.g. leukemia and lymphoma) can cause hyperuricemia → uric acid stones
- 10. **Falling shards**: tumor lysis syndrome can cause hyperuricemia and uric acid stones
- 11. **Mysterious acid secretions**: idiopathic acidic urine (50% of patients with uric acid stone)
- 12. **Acid puddles**: uric acid stones preferentially form in an acidic urine environment
- 13. Draining mud : metabolic acidosis (e.g. caused by chronic diarrhea)
 → increased H+ excretion → uric acid stones
- 14. **Dumping alkaline fluid**: alkalinization of the urine (e.g. with potassium citrate, potassium bicarb) can effectively treat and prevent uric acid stones
- Pure nun: allopurinol can prevent uric acid stones (with high cell turnover, or high purine metabolism)
- 16. Diamond windows: uric acid stones have characteristic diamondshaped, yellow-brown crystals on microscopy
- 17. "Cistern": cystine stones (occur in cystinuria)

- Acid pools: cystine stones form preferentially in an acidic urine environment
- COAL: cystinuria is caused by a defect in PCT reabsorption of cystine, ornithine, arginine, and lysine
- 20. Receding shy kid: cystinuria is caused by an autosomal recessive defect of resorption of amino acids in the PCT
- 21. **Kids carrying coal**: cystinuria usually presents in childhood with recurrent, non-calcium, renal stones
- 22. Stag antler: cystine stones can also present as "staghorn" calculi
- 23. **Hexagonal coal briquettes**: cystine crystals have a characteristic hexagonal shape on microscopy
- 24. **Blue "Nitro-pressure" smoke**: the diagnostic test for cystinuria is the sodium cyanide-nitroprusside urine test
- 25. **Pink stream in the smoke**: urine of a patient with cystinuria will turn red-purple in a positive sodium cyanide-nitroprusside test
- 26. **Pencil MINE**: in severe cases, penicillamine can be used to chelate and lower cystine levels (prevent cystine stones)
- 27. Grasping crane: chelating agent (penicillamine)
- 28. **Translucent X-ray flag on church**: uric acid and cystine stones are radiolucent (Calcium and MAP stones are radiopaque)
- 29. **Translucent X-ray flag on COAL mine**: uric acid and cystine stones are radiolucent (Calcium and MAP stones are radiopaque)



- "RENASCCAR": RENAL CELL CARCINOMA (RCC) (most common malignant renal tumor)
- 2. Older male: RCC is most common in men in 6th to 7th decade of life
- 3. Steaming angry: HYPERTENSION is a risk factor for RCC
- 4. Obese belly: OBESITY is a risk factor for RCC
- 5. Smoker: SMOKING is a risk factor for RCC
- 6. Clear cellular pattern : CLEAR CELL carcinoma (most common type of RCC)
- 7. Donuts & Candy : CLEAR CELL RCC are composed of cells with high LIPID & GLYCOGEN content \rightarrow large CLEAR VACUOLATED cytoplasm
- 8. "Pro Car Track": CLEAR CELL RCC arises from cells of PROXIMAL CONVOLUTED TUBULE
- "VHL" decal: CLEAR CELL RCC is frequently caused by mutations in VON HIPPEL LINDAU gene on chromosome 3 (in both SPORADIC and HEREDITARY cases)
- 10. 2 pink cancer fighting ribbons: VHL is a tumor SUPPRESSOR gene (cancer forms only if BOTH alleles are mutated) (both SOMATIC mutations in SPORADIC tumors)
- 11. "VHL" twins: VHL SYNDROME (FAMILIAL cancer syndrome) follows "2 hit" model (one VHL allele has an inherited GERMLINE mutation → cancer occurs if second allele develops a SOMATIC mutation during lifetime)
- 12. Red hair bun & curl over sunglasses: VHL SYNDROME predisposes to HEMANGIOBLASTOMAS of cerebellum & retina
- 13. **LUMPY yellow helmet**: CLEAR CELL RCC forms large, UNILATERAL WELL-DEFINED yellow-grey tumors with satellite NODULES
- 14. 2 lumpy yellow helmets: in VHL SYNDROME, CLEAR CELL RCC is often BILATERAL or multifocal
- 15. Chasing up blue pole : RCC can invade the RENAL VEIN \rightarrow extend into INFERIOR VENA CAVA and right heart
- 16. Puffy blue pant legs, in RCC, tumor extension into IVC can cause venous congestion → back pressure leads to lower extremity edema, ascites, hepatic dysfunction :
- 17. Mass of dilated blue loops on LEFT side : RCC can present with LEFT sided VARICOCELE (tumor invasion or thrombosis of L renal vein → backup of blood into L TESTICULAR vein)
- 18. Hit in the side: RCC presents with FLANK PAIN
- 19. Spurting red stream: RCC presents with PAINLESS HEMATURIA
- 20. Full pockets: RCC presents with PALPABLE ABDOMINAL MASS
- 21. "AIR PRO": RCC can produce ERYTHROPOIETIN
- 22. Extra red-disc balloons: RCC can lead to POLYCYTHEMIA (paraneoplastic syndrome due to ectopic erythropoietin production by tumor cells)
- 23. **PthD scientist**: RCC can produce Parathyroid Hormone Aberrant Peptide (PTHrP)]

- 24. RAIN umbrella; RCC can produce RENIN (leads to paraneoplastic syndrome) :
- 25. ACTHion clapper board : RCC can produce an ACTH-like hormone (leads to a paraneoplastic syndrome)
- 26. Blood splatter: RCC metastasizes early (hematogenously)
- 27. Crab logos on chest: RCC commonly metastasizes to lungs
- 28. Crab logos on leg bones: RCC commonly metastasizes to bones
- 29. "Will Call": WILMS TUMOR (most common primary renal cancer in children)
- 30. Blast: WILMS TUMOR (alternately NEPHROBLASTOMA) derives from METANEPHRIC BLASTEMA (embryonal structure that gives rise to kidney)
- 31. "11": WILMS tumor is commonly caused by loss of function mutations in WT1 or WT2, both on chromosome 11
- 32. Young kid: WILMS tumors are usually diagnosed before age of 10
- 33. One well-encapsulated helmet : WILMS tumor is solitary, unilateral, and encapsulated
- 34. Happy with full pockets: WILMS tumor presents with ASYMPTOMATIC ABDOMINAL MASS
- 35. **Steaming angry**: WILMS tumor can cause HYPERTENSION (due to excess renin production)
- 36. RAIN umbrella: WILMS tumor can produce excess RENIN
- 37. Cancer posters: WILMS tumor is associated with HEREDITARY cancer syndromes (although most commonly SPORADIC)
- 38. "1 ticket at a time": WAGR syndrome & DENYS-DRASH syndrome are caused by abnormal WT-1 genes
- 39. Losing 1 of 2 pink ribbons: WAGR syndrome & DENYS-DRASH syndrome follow "two-hit" model (one allele has inherited germline mutation, cancer occurs if second allele develops somatic mutation during lifetime)
- 40. "Wagers": WAGR syndrome (presents with WILMS tumor, aniridia [absent iris], genitourinary malformations, mental retardation)
- ": DENYS-DRASH syndrome (presents with WILMS tumor, gonadal dysgenesis or male pseudohermaphroditism, early onset renal failure)
- 42. Ink running out on "Beck Wide-Win" poster: BECKWITH-WIEDEMANN syndrome is due to abnormal genomic imprinting at the WT2 locus
- "Beck WIDE-Win": BECKWITH-WIEDEMANN syndrome (presents with WILMS syndrome and "WIDE" features (macroglossia [enlarged tongue], enlarged body organs, omphalocele))
- 44. Red ketchup, meat, and fatty crust: Renal ANGIOmyoLIPOMA (benign kidney tumor)
- 45. Sclerotic tubers: RENAL ANGIOmyoLIPOMAS are common in TUBEROUS SCLEROSIS

Renal Pathology 6.1 Osmolality & Sodium Disorders



- 1. Big wet window pane : % of total body water and solutes are found in the intracellular compartment
- 2. Smaller right sided pane: ½ of total body water and solutes are found in the extracellular compartment (made of the interstitial and vascular spaces)
- 3. **Red quarter pane**: the vascular space contains about 25% of the total extracellular fluid (the remaining 75% is interstitial
- 4. **Bananas INSIDE the window**: the major INTRAcellular cation is potassium (with proteins and phosphate as conjugate anions
- 5. **Tray of peanuts in smaller window pane**: the major EXTRAcellular cation is sodium (with chloride and bicarbonate as conjugate anions)
- 6. Water dripping between compartments: water moves freely throughout the fluid compartments of the body until it is at equilibrium (based on osmotic pressure)
- condOsm-ment ketchup: osmolality (determined by the concentration of dissolved solutes) is the driving force for fluid movement between fluid compartments of the body (measured in mOsm)
- 8. Candied peaNats: sodium (and, less commonly, glucose or urea) is the primary solute determining serum osmolality
- 9. "2x the Nats!": since each sodium ion (the major extracellular cation) will have a conjugate anion, serum osmolality can be estimated by doubling serum sodium concentration
- 10. **"290 calories"** : Under normal conditions, serum osmolality is around 290 mOsm (285-295 mOsm)
- 11. **Green arrow by condOsment ketchup**: changes in OSMOLALITY are represented by changes in vertical window height (higher osmolality→ taller window, lower osmolality→ shorter window)
- 12. **PeaNats by osmolality arrow**: sodium is ALMOST ALWAYS the main determinant of plasma osmolality (hypernatremia→ increased osmolality, hyponatremia→ decreased osmolality)
- 13. **Volume bucket on right of platform**: changes in extracellular and intracellular VOLUME (determined by serum osmolality) are shown by extending the x-axis (HYPERvolemia) or decreasing the x-axis (HYPOvolemia)
- 14. **SECRETEly dumping peanut-filled brown liquid**: SECRETORY diarrhea (bacterial toxins, VIPoma) causes loss of large amounts of solute and water in stool→ loss of ISOtonic fluid→ sodium loss EQUALS water loss
- 15. **SECRETEly dumping peanut-filled brown liquid**: SECRETORY diarrhea (bacterial toxins, VIPoma) causes loss of large amounts of solute and water in stool→ loss of ISOtonic fluid→ sodium loss EQUALS water loss
- 16. **Opening window shifts small pane to left**: loss of ISOtonic fluid (secretory diarrhea) causes a decrease in EXTRAcellular fluid volume

- 17. **No change in larger pane**: loss of ISOtonic fluid contracts the EXTRAcellular space, but because there is no change in serum sodium or osmolality, INTRAcellular volume is unchanged
- 18. **NORMO bathroom window height** : loss of ISOtonic extracellular fluid→ no change in serum sodium or osmolality→ no change in INTRAcellular volume→ HYPOvolemic NORMOnatremia
- 19. **ISO IV bag on plant trellis** : gain of ISOtonic fluid (water gain matches sodium gain) expands the EXTRAcellular space→ no change in serum sodium or osmolality→ INTRAcellular volume is unchanged
- 20. **Trellis extends window to right**: ISOtonic expansion of the EXTRAcellular compartment increases interstitial fluid (increased intravascular hydrostatic pressure→ extravasation of intravascular fluid to interstitium
- 21. **Happy lady in window pane** : gain of ISOtonic fluid expands the EXTRAcellular compartment (especially the interstitium) → no change in serum sodium or osmolality → INTRAcellular volume is unchanged
- 22. **Balcony extended to right**: gain of ISOtonic fluid (normal saline)→ increase in EXTRAcellular volume with no change in INTRAcellular volume→ HYPERvolemic NORMOnatremia
- 23. **THIGH-azide thighs by ADH cooler** : diuretics \rightarrow volume depletion and ADH release (free water reabsorption) \rightarrow loss of SODIUM greater than water
- 24. **Torn adrenal hat by ADH cooler** : adrenal insufficiency (hypoaldosteronism) → volume depletion and ADH release→ loss of SODIUM greater than water
- 25. Man pushing window screen down : loss of SODIUM greater than water→ decreased serum sodium → DECREASED osmolality→ movement of water from intracellular space to extracellular space
- 26. **Window screen shifted down AND to the left** : loss of EXTRAcellular AND INTRAcellular volume due to DECREASED serum sodium and osmolality → HYPOvolemic HYPOnatremia
- 27. ADH cooler behind clothesline covering window : syndrome of inappropriate ADH (SIADH) causes gain of FREE WATER ONLY→ decreased serum sodium → DECREASED serum osmolality
- 28. Kidney shaped sock leaking water onto arrow: with normal renal function, increased urine output can maintain EUvolemia (even in SIADH)
- 29. **Normal horizontal clothesline covering window**: increased free water→ decreased serum sodium→ DECREASED osmolality with NORMAL volume status→ EUvolemic HYPOnatremia

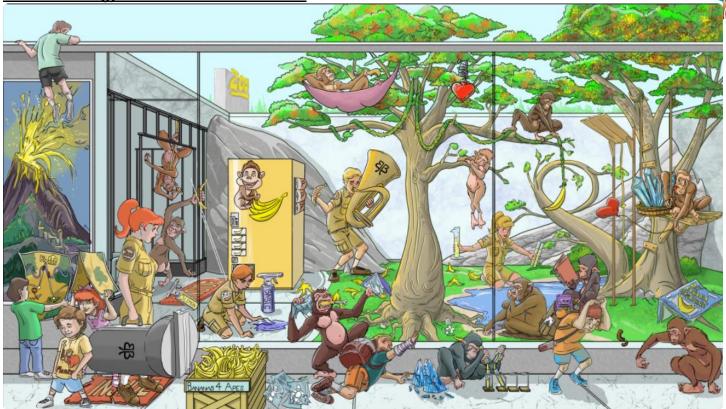
Renal Pathology 6.1 Osmolality & Sodium Disorders





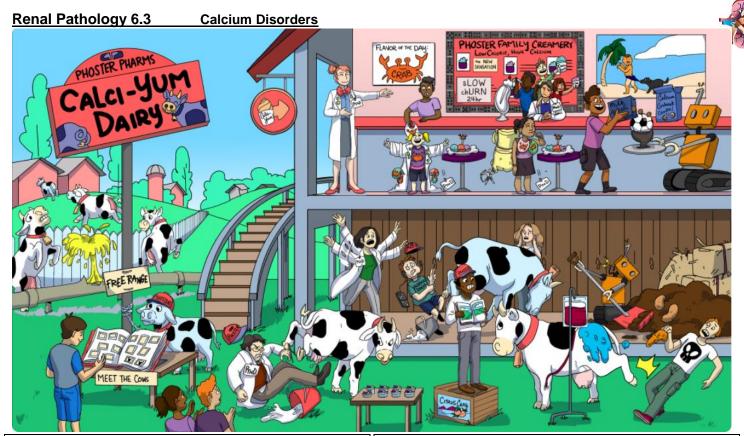
- 30. Floppy heart balloon, liver coral, and shipwrecked kidney with ADH free water pump and mineralocorticoid crystal: conditions of RELATIVE hypovolemia (decreased intravascular volume with increased total body volume) cause activation of the renin-angiotensin and ADH systems→ gain of WATER greater than sodium
- 31. **ADH free water pump on top** : constitutive release of ADH (from intravascular hypovolemia) → gain of WATER greater than sodium
- 32. **Expanded tank to left and right**: gain of WATER greater than sodium→ decreased serum sodium→ DECREASED serum OSMOLALITY→ movement of water into INTRAcellular space→ expansion of BOTH intracellular and extracellular space
- 33. Floppy heart balloon : hypotension from decreased cardiac output→ constitutive activation of renin-angiotensin and ADH systems → chronic volume retention→ CHRONIC HYPERvolemia
- 34. **Stony liver coral** : cirrhosis impairs albumin production→ low serum oncotic pressure→ INTRAVASCULAR volume depletion due to fluid extravasation to interstitial space (ascites, edema)
- 35. **Shipwrecked cracked kidney**: nephrotic syndrome causes loss of albumin in the urine→ low serum oncotic pressure→ INTRAVASCULAR volume depletion due to fluid extravasation to interstitial space (edema)
- 36. Tank stretched to left and right covering window : expansion of BOTH the intracellular and extracellular compartments with DECREASED osmolality→ HYPERvolemic HYPOnatremia
- 37. Nauseated on top floor : vomiting causes loss of WATER greater than sodium— increased serum sodium— INCREASED serum osmolality
- 38. **Spilling sugary chocolate cereal from tall window**: OSMOTIC diarrhea (laxatives or lactulose) causes loss of WATER greater than sodium→ increased serum sodium→ INCREASED serum osmolality
- 39. **Spilling sugary yellow drink from tall window**: osmotic diuresis (hyperglycemia) causes loss of WATER greater than sodium→ increased serum sodium→ INCREASED serum osmolality
- 40. Nauseated man in tall window: vomiting, OSMOTIC diarrhea, and osmotic diuresis cause a loss of WATER greater than sodium \rightarrow increased serum OSMOLALITY
- 41. Nauseous kid with both curtains drawn: loss of HYPOtonic fluid from the extravascular space→ INCREASED serum osmolality→ movement of water from intracellular to extracellular space→ contraction of BOTH intracellular and extracellular space
- 42. **Curtains drawn in tall window**: contraction of both the intracellular and extracellular space due to INCREASED serum osmolality (vomiting, osmotic diuresis, sweating)→ HYPOvolemic HYPERnatremia
- 43. **Sweating by desert cactus**: both profuse sweating and water restriction lead to loss of WATER greater than sodium→ increased serum osmolality→ contraction of BOTH intracellular and extracellular compartments→ HYPOvolemia HYPERnatremia

- 44. **Sweating kid in cherub bath with barely drawn curtains**: free water loss (sweating or diabetes insipidus) → increased serum osmolality→ movement of water from intracellular compartment to extracellular compartment→ contraction of both INTRAcellular and EXTRAcellular compartments
- 45. **Kid in tall window drinking water is happy**: thirst (stimulated by increased serum osmolality) is usually powerful enough to maintain volume status in the face of free water loss→ EUvolemic HYPERnatremia
- 46. **Salty sodium peanuts in bird feeder**: administration of hypertonic saline, sodium bicarbonate, or antibiotics with sodium conjugates can lead to MASSIVE sodium overload
- 47. **Tall window by bird feeder** : SODIUM gain greater than water (as with hypertonic saline administration) \rightarrow increased serum sodium \rightarrow INCREASED serum osmolality
- 48. **Guy pulling curtain toward bird feeder on right**: administration of excess sodium→ increased serum OSMOLALITY→ movement of water from intracellular to extracellular space→ EXPANSION of extracellular space with CONTRACTION of intracellular space→ HYPERvolemic HYPERnatremia
- 49. **Tall window with guy eating candy**: glucose is osmotically active and in states of marked hyperglycemia (such as DKA or HHS in diabetes mellitus) serum osmolality is INCREASED despite normal serum sodium
- 50. Curtains drawn tightly around candy race: increased serum osmolality (due to hyperglycemia) draws water OUT of the intracellular space while osmotic diuresis (due to renal excretion of glucose) draws water out of extracellular space—severe contraction of BOTH the intracellular and extracellular compartments—HYPOvolemic HYPEROSMOLALITY
- 51. **Spilled yellow drinks knocks over peanuts**: increased serum osmolality (from marked hyperglycemia) draws water from the intracellular space into the extracellular space → dilution of serum sodium concentration → FALSELY normal or low serum sodium level
- 52. **Guy holding head near fallen peanuts**: symptoms of hyponatremia include confusion, altered mental status, and lethargy
- 53. **Guy by fallen peanuts about to vomit**: symptoms of hyponatremia include nausea and vomiting
- 54. **Bucket of water falling on head by low windows**: severe cases of hyponatremia can cause cerebral edema (decreased extracellular osmolality→ shift of water INTO neurons)→ seizures and coma
- 55. **Man locked in cell by flying**: correcting hyponatremia too quickly→ rapid movement of water out of cells→ breakdown of myelin (in an attempt to raise INTRAcellular osmolality and maintain volume) → severe neurologic dysfunction (osmotic demyelination syndrome)
- 56. **Stuck in jail with eyes wide open**: osmotic demyelination of pontine white matter→ paralysis of all voluntary movement except of the eyes and eyelids ("locked in syndrome")



- 1. Bananas 4 apes : normal serum potassium is around 4 mEq/L (3.5-5 mEq/L)
- 2. Depleted banana peels : hypokalemia is defined as a serum potassium < 3.5 mEg/L
- 3. **High dive to loop de loop**: loop and thiazide diuretics (non-K sparing diuretics) → increased sodium delivery to collecting ducts→ increased Na/K exchange→ potassium EXCRETION and hypokalemia
- 4. **Mineralocorticoid crystals**: PRIMARY hyperaldosteronism (Conn syndrome) or diseases with increased mineralocorticoid ACTIVITY (Cushing's syndrome, CAH) cause increased potassium excretion in the collecting ducts→hypokalemia
- 5. **Floppy heart balloon**: SECONDARY hyperaldosteronism (CHF, cirrhosis, nephrotic syndrome) → activation of the renin-angiotensin-aldosterone system due to RELATIVE hypovolemia→ potassium wasting→ hypokalemia
- 6. Zookeeper with "1" and "II" acid test tubes : renal tubular acidosis type 1 and 2 cause potassium wasting→ hypokalemia
- Fallen banana Magazine: hypomagnesemia can inhibit potassium absorption → hypokalemia (refractory without correction of hypomagnesemia)
- 8. **Sick monkey in brown puddle**: diarrhea leads to loss of potassium-rich intestinal secretions→ hypokalemia (less common with vomiting gastric fluid)
- 9. Running over INSIDE mat into cell : insulin causes a shift of potassium INTO cells→ lower SERUM potassium with normal TOTAL potassium
- 10. **Beta bugle tuba**: beta-2 agonists (albuterol) stimulate a shift of potassium INTO cells→ lower SERUM potassium with normal TOTAL potassium
- 11. **3 "P"** batteries in banana vending machine: insulin and beta-2 agonists (albuterol) cause a shift of potassium INTO cells by stimulating Na/K-ATPases (which pump 2 potassium ions into cells for every 3 sodium ions pumped out)
- 12. "BASIC" ape poop cleaner : alkalosis causes a shift of potassium INTO cells in exchange for hydrogen ions (to help buffer alkalosis and lower serum pH) → REDUCED serum potassium in alkalosis
- 13. **Falling "LEMON" cleaner**: vomiting, diuretics, and hyperventilation can all lead to reduced acid levels (alkalosis) \rightarrow INTRAcellular potassium shift \rightarrow lower serum potassium
- 14. **Weak monkey climbing**: hypokalemia can lead to muscle weakness, decreased deep tendon reflexes, and ascending paralysis (severe cases)
- 15. Ape stepping on EKG vine: hypokalemia can cause flat or inverted T waves on EKG
- 16. Vine pulling banana Up: hypokalemia can cause U waves on EKG (small positive inflections following the T wave)

- 17. **Heart spring in tree**: hypokalemia can cause cardiac conduction abnormalities including supraventricular tachycardia, ectopic beats, and even VT/VF
- 18. **Kidney shaped poop**: chronic kidney disease decreases potassium filtration and excretion→ hyperkalemia
- 19. **ACE phone cover** : ACE inhibitors and ARBs inhibit the action of aldosterone→ decreased potassium excretion→ hyperkalemia
- 20. Spiral notebook→ spironolactone (K sparing diuretic) antagonizes aldosterone receptors→ decreased potassium excretion→ hyperkalemia :
- 21. Almonds and tangerine: amiloride and triamterene (ENaC inhibitors) decrease potassium excretion→ hyperkalemia
- 22. **Broken mineralocorticoid crystal** : HYPOaldosteronism (Addison's disease, adrenal insufficiency)→ decreased potassium excretion→ hyperkalemia
- 23. Four acid tubes forming "K" : type 4 renal tubular acidosis leads to decreased potassium excretion→ hyperkalemia
- 24. **Trauma cast**: tissue damage following trauma can cause massive cell death→ release of intracellular potassium→ hyperkalemia
- 25. **Chomping chicken muscle meat**: rhabdomyolysis (from trauma, medications, or inflammatory myopathies) involves massive muscle breakdown and cell death→ release of intracellular potassium→ hyperkalemia
- 26. **Tearing lines from story** : chemotherapy causes extensive tumor cell death→ release of intracellular potassium→ hyperkalemia
- 27. **Broken INSIDE mat**: insulin deficiency (type I diabetes) decreases cellular potassium uptake by inhibiting Na/K-ATPase activity→ hyperkalemia
- 28. **Blocking beta-2 tuba case** : beta-2 blockers inhibit cellular potassium uptake by inhibiting Na/K-ATPase activity→ hyperkalemia
- 29. **Acid volcano mural** : acidosis causes a shift of potassium OUT of cells in exchange for hydrogen ions (to help buffer acidosis and raise serum pH) → INCREASED serum potassium in acidosis
- 30. "T" shaped map: tall, "peaked" T waves are the earliest EKG finding in hyperkalemia
- 31. **Sine wave on map**: severe hyperkalemia can cause widening of the QRS complex→ "sine wave" EKG appearance
- 32. **Shaking heart on tee shirt**: severe hyperkalemia can lead to complete heart block, VF/VT, torsades de pointes, or asystole
- 33. Too weak to climb wall: hyperkalemia can lead to muscle weakness, decreased deep tendon reflexes, and ascending paralysis (severe cases)



- 1. "Meet the Cows" album : around 45% of serum calcium circulates bound to albumin
- 2. **Phoster farm cow hat**: around 5% of serum calcium circulates bound to anions such as phosphate
- 3. "FREE Range" cows: around 50% of serum calcium circulates in its IONIZED or "FREE" form (the metabolically active form of calcium)
- 4. Cow jumped over the acid pipe: acidosis causes albumin to release bound calcium (to bind hydrogen ions and buffer acidosis) \rightarrow increased serum IONIZED calcium \rightarrow signs of hypercalcemia
- 5. **"9" cow tail** : normal total serum calcium is 8.5-10.2 mg/dL (around 9 mg/dL)
- 6. Falling PThD spilling milk: low levels of PTH in hypoparathyroidism (primary hypoparathyroidism)— decreased calcium absorption in the intestines and kidneys hypocalcemia
- 7. **Phlying Phoster hat**: low PTH in hypoparathyroidism causes hyperphosphatemia (PTH normally stimulates calcium reabsorption and phosphate EXCRETION in the kidney
- 8. Albright scholar's domino phone case and G headphones: Albright hereditary osteodystrophy (pseudohypoparathyroidism type 1A) is due to an autosomal dominant mutation affecting a G-protein involved in PTH receptor signaling
- PThD unsuccessfully trying to signal Albright scholar: abnormal PTH receptor signaling→ PTH RESISTANCE in Albright hereditary osteodystrophy
- 10. **Albright scholar spilling milk**: PTH resistance in Albright hereditary osteodystrophy→ hypocalcemia (decreased absorption of calcium in intestines and kidneys)
- 11. **Albright scholar flying Phoster hat**: PTH resistance in Albright hereditary osteodystrophy→ hyperphosphatemia (decreased excretion of phosphate in kidneys)
- 12. **Chunky Albright scholar's fingers**: characteristics of Albright hereditary osteodystrophy include shortened 4th and 5th digits, short stature, and obesity

- 13. **Albright scholar's scared mom**: Albright hereditary osteodystrophy is inherited from mothers ONLY due to genetic imprinting (the expression of certain genes from only one parent)
- 14. **Broken CALCITRON robot**: deficiency of vitamin D (which normally increases calcium absorption in the intestines and kidneys) is a common cause of hypocalcemia
- 15. **Shriveled kidney-shaped poops**: chronic kidney disease results in 1-alpha hydroxylase deficiency→ decreased hydroxylation of 25-hydroxyvitamin D to 1,25-dihydroxyvitamin D (biologically active vitamin D) → vitamin D and calcium deficiency
- 16. Citrus crate by IV bag: citrate (a preservative in stored blood) can bind calcium and cause hypocalcemia with blood transfusion
- 17. **Phoster hat by IV bag** : phosphate in transfused blood products can bind calcium— hypocalcemia
- 18. Photo album by IV bag : albumin in blood products or albumin concentrates can bind calcium \rightarrow hypocalcemia
- 19. **Pill cups**: foscarnet, loop diuretics, bisphosphonates, and cinacalcet are all associated with hypocalcemia
- 20. Pancreas sponge and skull shirt: severe acute pancreatitis causes extensive fat necrosis and release of fatty acids, which bind large amounts of calcium→ dystrophic calcification and hypocalcemia



- 21. **High Calci-YUM sign**: hypercalcemia is defined as a TOTAL serum calcium >10.5 mg/dL or an IONIZED calcium > 1.4 mg/dL
- 22. **PThD with 4 lapel buttons around bowtie**: PRIMARY hyperparathyroidism (HYPERcalcemia with HIGH PTH) is responsible for over 95% of cases of hypercalcemia (usually due to parathyroid adenoma)
- 23. Cancer crab ice cream sign: malignancy is the second most common cause of hypercalcemia (following primary hyperparathyroidism) and tends to be more severe
- 24. **Pretending to be PThD**: hypercalcemia of malignancy can be due to production of ectopic parathyroid hormone RELATED protein, which mimics the action of normal PTH
- 25. Cancer crab bibs over chest and pockets: paraneoplastic PTH related protein is most commonly produced by squamous cell lung cancer and renal carcinoma→ hypercalcemia of malignancy
- 26. PThD tag falling under cancer crab ice cream: hypercalcemia of malignancy caused by PTH related protein leads to HIGH calcium, HIGH PTHrP, and LOW normal PTH (parathyroids are suppressed due to hypercalcemia)
- 27. Lytic scoops out of crab ice cream : hypercalcemia of malignancy can be due to osteoLYTIC bone metastases→ bone breakdown and activation of osteoclasts by tumor-released humoral factors
- 28. Cancer crab bib girl wearing vertebral backpack: osteoLYTIC bone metastases are common in areas of active hematopoiesis (vertebrae, sternum)
- 29. **Pink cancer crab bib over chest**: breast and lung cancer often cause lytic bone lesions→ hypercalcemia of malignancy
- 30. **B-cell archer keychain**: multiple myeloma (malignancy of plasma cells) often causes lytic bone lesions→ hypercalcemia of malignancy
- 31. Familial "Low Calorie, High Calcium" ice cream: patients with familial hypocalciuric hypercalcemia have abnormally LOW calcium excretion in the URINE and abnormally HIGH calcium in the BLOOD
- 32. **Domino pattern around Family Creamery sign**: familial hypocalciuric hypercalcemia is inherited in an autosomal dominant fashion

- 33. **New SENSATION**: familial hypocalciuric hypercalcemia is due to autosomal dominant mutation of the calcium sensing receptors (CaSR) in the kidneys and parathyroid glands
- 34. **Kidney shaped milkshake** : abnormal calcium sensing in the kidneys→ inappropriate reabsorption of calcium→ HYPOcalciuria and HYPERcalcemia
- 35. **Too many calcium kids on PThD**: abnormal calcium sensing in the parathyroid glands raises the calcium threshold→ increased PTH release (even with hypercalcemia)
- 36. **sLOW chURN 24hr**: familial hypocalciuric hypercalcemia is diagnosed by measuring LOW 24 hour calcium excretion with HIGH serum calcium (as opposed to increased urinary calcium excretion in primary hyperparathyroidism)
- 37. Pale THIGHS : thiazide diuretics increase calcium reabsorption in the distal tubule \to increased serum calcium
- 38. Cookies n' cream soccer ball scoop : vitamin D toxicity can be caused by ingestion or granulomatous diseases such as sarcoidosis (in which macrophages produce excess vitamin D) \rightarrow hypercalcemia
- 39. **Milk alkali malt balls with calcium carbonate center**: milk alkali syndrome following ingestion of calcium-containing antacids (such as calcium carbonate) consists of hypercalcemia, metabolic alkalosis, and acute kidney injury

Renal Pathology 6.4 Acid/Base Disorders Disorders

Acid/Base Disorders

Acid/Base Disorders

- 1. **pHrodo** : acid/base disorders
- 2. LOW where acid lava flows: ACIDOSIS (pH < 7.35)
- 3. HIGH blue basic pools: ALKALOSIS (pH > 7.45)
- 4. GAPwise stuffing his face LOW in scene: METABOLIC ACIDOSIS
- 5. LOW where acid lava flows: ACIDOSIS (pH < 7.35)
- 6. **FALLING bicarb baking soda**: in Metabolic Acidosis, serum BICARBONATE is LOW (< 21 mEq/L)
- 7. Carbon fumes LOW to the ground : in Metabolic Acidosis, PaCO2 is LOW (< 35 mmHg) (due to compensatory respiratory alkalosis [lungs increase ventilation \rightarrow blow off CO2 \rightarrow increases pH CLOSER to normal)
- 8. None sHall Cross: ANION GAP = Na HCO3 CI
- 9. "12" pack supplies: INCREASED Anion Gap Metabolic Acidosis (anion gap > 12) (caused by excessive unmeasured organic acids [MUDPILES])
- 10. "**M" liquid**: METHANOL causes Increased Anion Gap Metabolic Acidosis (windshield washer fluid, sterno, paint solvents) (metabolized by alcohol dehydrogenase into formic acid → damages optic nerve)
- 11. **BUN bag**: UREMIA (increased blood urea nitrogen [BUN]) is associated with Increased Anion Gap Metabolic Acidosis (decreased kidney function → decreased filtration of organic acids and other waste products → increased serum uric acid, sulfuric acid, phosphoric acid)
- 12. **Candy KEY**: DIABETIC KETOacidosis (DKA) causes Increased Anion Gap Metabolic Acidosis (due to accumulation of ketones [organic acids])
- 13. "P" liquid: PROPYLENE GLYCOL causes Increased Anion Gap Metabolic Acidosis (antifreeze) (converted via alcohol dehydrogenase into acid)
- 14. **Rusty "Isolation canyon"**: acute IRON poisoning causes Increased Anion Gap Metabolic Acidosis (ferric ions become hydrated and release protons)
- 15. **Acidic milk**: LACTIC ACID causes Increased Anion Gap Metabolic Acidosis (hypoxia → anaerobic metabolism → lactic acid production)
- 16. **"E" liquid**: ETHYLENE GLYCOL causes Increased Anion Gap Metabolic Acidosis (antifreeze) (metabolized by alcohol dehydrogenase into acid)
- 17. **ASA umpire armour**: excessive SALICYLIC acid (aspirin) causes Increased Anion Gap Metabolic Acidosis (directly as an organic acid AND via production of lactic acid [ASA → mitochondrial injury → tissue hypoxia)

- 18. Osm = 2Na +glucose/18 + BUN/2.8 + EtOH/4.6 : calculation for serum OSMolality
- 19. "10" axe & shield : INCREASED OSMole gap (ACTUAL osmolality CALCULATED osmolality > 10) indicates presence of unaccounted osmoles
- 20. **kEtchuP Mustard condOSMents**: ETylene glycol, Propylene glycol & Methanol are active OSMoles (increase osmole gap)
- 21. **Elevated chloride crystals**: NON-anion gap Metabolic Acidosis is HYPERchloremic (LOW bicarb is replaced by INCREASING CHLORIDE in order to maintain isoelectricity
- 22. **Brown puddle**: excessive DIARRHEA causes NON-anion gap Metabolic Acidosis (due to loss of bicarb from pancreatic secretions)
- 23. Renal tubule vial : Renal Tubular Acidosis (RTA) causes NON-anion gap Metabolic Acidosis
- 24. SeaGASP sitting LOW in scene : RESPIRATORY ACIDOSIS
- 25. LOW where acid lava flows : ACIDOSIS (pH < 7.35)
- 26. Carbon fumes rising HIGH : in Respiratory Acidosis, PaCO is HIGH (> 45 $\mbox{mmHg})$
- 27. **Raised bicarb baking soda**: in Respiratory Acidosis, serum BICARB is HIGH (> 27 mEq/L) (due to compensatory metabolic alkalosis [kidneys increase bicarb retention and production → increases pH CLOSER to normal])
- 28. **Blue bloater**: COPD can cause Respiratory Acidosis (decreased respiratory drive & impaired gas exchange → chronic CO2 retention)
- 29. **Snore lines** : Obstructive Sleep Apnea can cause Respiratory Acidosis (obstruction → CO2 retention)
- 30. Sleeping potion : Opioids & other Sedatives can cause Respiratory Acidosis (CNS depression \rightarrow decreased respiratory drive \rightarrow CO2 retention)
- 31. **Lock around chest** : Restrictive Lung Disease can cause Respiratory Acidosis (pulmonary fibrosis, scoliosis, obesity, pleural effusion → CO2 retention)
- 32. **Neuronal dangly bits**: Neurological Disorders can cause Respiratory Acidosis (ALS, Guillain-barre, polio, myasthenia gravis, botulism) (interrupted nerve transition to diaphragm → CO2 retention)
- 33. Atrophied muscles : Muscle Disorders can cause Respiratory Acidosis (muscular dystrophy) (diaphragm too weak to ventilate \rightarrow CO2 retention)

Renal Pathology 6.4 Acid/Base Disorders Disorders



- 34. Stuffing face HIGH in blue pools: METABOLIC ALKALOSIS
- 35. HIGH blue basic pools: ALKALOSIS (pH > 7.45)
- 36. Pools near raised bicarb baking soda : in Metabolic Alkalosis, serum BICARB is HIGH (>27 mEq/L)
- 37. Blue pool near HIGH carbon fumes : in Metabolic Alkalosis, PaCO is HIGH (> 45 mmHg) (due to compensatory respiratory acidosis [lungs decrease ventilation \rightarrow CO2 retention \rightarrow decreases pH CLOSER to normal
- 38. **Vomit**: VOMITING can cause Metabolic Alkalosis (loss of HCl from stomach → retention of BICARB)
- 39. **Diuresis**: DIURETICS can cause Metabolic Alkalosis (loop & thiazide diuretics → excretion of H+ in exchange for Na+ → retention of BICARB)
- 40. IV backpack & deploying CHLORIDE crystal BOULDER: in CHLORIDE SENSITIVE alkalosis, a BOLUS of normal saline (contains CHLORIDE) corrects hypovolemia → CORRECTS metabolic alkalosis (if caused by vomiting or diuretics)
- 41. **Oversized MINERALocorticoid crystal**: Mineralocorticoid excess can lead to Metabolic Alkalosis (ALDOSTERONE leads to loss of H+ in the collecting duct → retention of BICARB) (chloride RESISTANT due to simultaneous increase in volume)

- 42. Hydroxyl bubble wand : RESPIRATORY ALKALOSIS
- 43. HIGH blue basic pools: ALKALOSIS (pH > 7.45)
- 44. **Waterfall near LOW carbon fumes** : in Respiratory Alkalosis, PaCO2 is LOW (< 35 mmHg)
- 45. **Waterfall near FALLING bicarb**: in Respiratory Alkalosis, serum BICARB is LOW (<21 mEq/L) (due to compensatory metabolic acidosis [kidneys increase bicarb excretion and decrease production → decreases pH CLOSER to normal])
- 46. **HIGH waterfall**: High Altitude can cause Respiratory Alkalosis (decreased O2 content \rightarrow hypoxemia \rightarrow peripheral chemoreceptors sense low PaO2 \rightarrow stimulate respiratory center to increase ventilation \rightarrow loss of CO2)
- 47. **Nest clot in pulmonary tree**: Pulmonary Embolism can cause Respiratory Alkalosis (hypoxemia and lung irritation \rightarrow activate respiratory centers to increase ventilation \rightarrow loss of CO2)
- 48. **Sweating anxiously**: Panic Attacks can cause Respiratory Alkalosis (hyperventilation → loss of CO2)
- 49. Inhaler : Asthma can cause Respiratory Alkalosis (hypoxemia, lung irritation & anxiety \rightarrow stimulate respiratory centers to increase ventilation \rightarrow loss of CO2)
- 50. ASA umpire disguise : Aspirin can cause Respiratory Alkalosis (directly stimulates respiratory centers to increase ventilation \to loss of CO2

Renal Pathology 6.5

Renal Tubular Acidosis (RTA)

- 1. "Acid Inspector" in Kidney Land: Renal Tubular Acidosis (RTA) (conditions that cause NON-anion gap Metabolic Acidosis due to malfunctioning renal tubules)
- 2. Pro Cart Track: Proximal Convoluted Tubule
- 3. Canoe Duct: Collecting Duct
- 4. Close Gap: RTA causes a NORMAL anion gap metabolic acidosis
- 5. INTERnational food truck near canoe duct : INTERCALATED cells of the collecting duct $\,$
- 6. "3P" truck pumping out yellow fluid : intercalated cells contain a H+ ATPase NORMALLY secretes protons (H+) into the collecting duct lumen
- 7. NH4+ acid disposal bucket : H+ secreted by the H+ ATP-ase leads to conversion of ammonia (NH3) into ammonium (NH4+) \rightarrow secretion of acid in the form of NH4+

Type 1 RTA

- 8. Inspecting 1 yellow test tube: type 1 (DISTAL) RTA
- 9. **Broken INTERnational food truck**: type 1 RTA is caused by DYSFUNCTIONAL H+ ATP-ase within intercalated cells
- 10. Yellow fluid seeping from truck: TYPE 1 RTA leads to METABOLIC ACIDOSIS (due to dysfunctional H+ ATPase)
- 11. Truck pumping out minimal yellow fluid : in type 1 RTA, SECRETION of H+ onto anions and ammonia is DECREASED (due to dysfunctional H+ ATPase)
- 12. "5" "5" canoe in blue water : in type 1 RTA, URINE is relatively BASIC (pH > 5.5) (dysfunctional H+ ATPase → decreased secretion of H+)
- 13. Persistently "NH3" bucket : in type 1 RTA, URINE ammonIUM (NH4+) is LOW (dysfunctional H+ ATPase \rightarrow decreased secretion of H+)
- 14. Falling bananas : type 1 RTA presents with HYPOKALEMIA (dysfunctional H+ ATPase \to K+ is secreted instead of H+)
- 15. **Stones with "P" fossils in blue river** : type 1 RTA can present with CALCIUM PHOSPHATE kidney stones (metabolic acidosis → increased bone resorption → excess calcium AND phosphate) (alkaline urine facilitates formation of calcium phosphate stones
- 16. **Broken "Citrus Crate" in blue river** : in type 1 RTA, URINE CITRATE is LOW (because citrate is used to buffer metabolic acidosis) → contributes to calcium stone formation (citrate normally binds calcium in urine)
- 17. **Antibody lights**: autoimmune disorders (Sjogren syndrome, rheumatoid arthritis) are the most common cause of type 1 RTA in adults)
- 18. **Dripping ice cream**: disorders that cause hypercalcemia (hyperparathyroidism, idiopathic hypercalciuria) can cause type 1 RTA
- 19. **Kid**: hereditary disorders are the most common cause of type 1 RTA in children
- 20. AMPHibian: amphotericin B can cause type 1 RTA
- 21. LIFTIUM: lithium can cause type 1 RTA

Renal Pathology 6.5

Renal Tubular Acidosis (RTA)

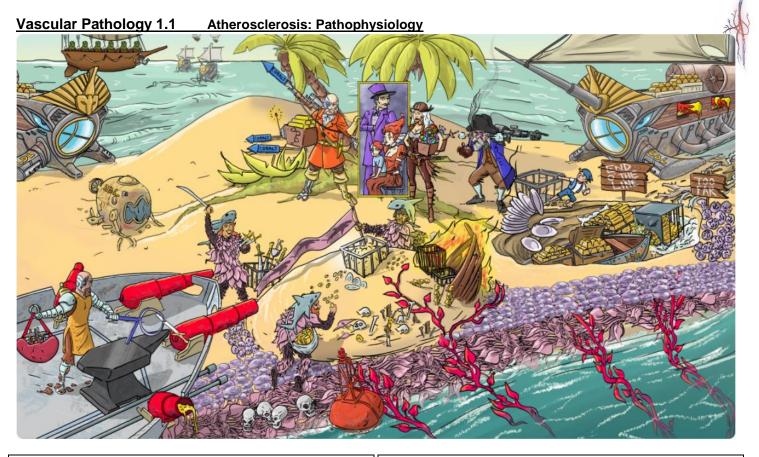
- 22. "Bicarb Bakery" cupcakes distributed near "PRO CART TRACK": bicarbonate is REABSORBED in the proximal tubule
- 23. CAR battery : CARbonic anhydrase (within proximal tubule lumen) converts bicarb into CO2 and water \rightarrow CO2 absorbed into cell \rightarrow converted back to bicarb within cell

Type 2 RTA

- 24. Inspecting 2 yellow test tubes: type 2 (PROXIMAL) RTA
- 25. **Tipping truck leading to bicarb wasting**: TYPE 2 RTA is caused by DECREASED bicarbonate REABSORPTION in the proximal convoluted tubule \rightarrow bicarb wasting in urine \rightarrow non-anion gap metabolic acidosis
- 26. Limited Edition, More Later: type 2 RTA is SELF-LIMITED (bicarb wasting → decreased serum bicarb → decreased filtration in nephron → DISTAL nephron is capable of reabsorbing the small amount of filtered bicarb)
- 27. Blue water pool: in type 2 RTA, URINE is initially relatively basic (pH > 5.5)
- 28. Blue puddle turning into yellow lemonade: in type 2 RTA, URINE eventually becomes a more normal pH (due to new equilibrium)
- 29. **Falling bananas**: type 2 RTA presents with HYPOKALEMIA (excessive bicarb in tubules binds Na+ \rightarrow sodium bicarb travels distally \rightarrow in collecting duct, Na+ is exchanged for K+ \rightarrow K+ wasting
- 30. Fan-Cone near "Pro Cart Track": Fanconi syndrome (generalized dysfunction of Proximal Tubule → type 2 RTA combined with wasting of phosphate, uric acid glucose. small proteins)
- 31. **Kid with Fan-Cone**: in children, Fanconi syndrome is most commonly due to an inherited enzyme disorder
- 32. Battery acid inhibiting car battery: acetazolamide can cause type 2 RTA (inhibits carbonic anhydrase \rightarrow bicarb in lumen is not converted into carbon dioxide and water \rightarrow no bicarb reabsorption in proximal tubule

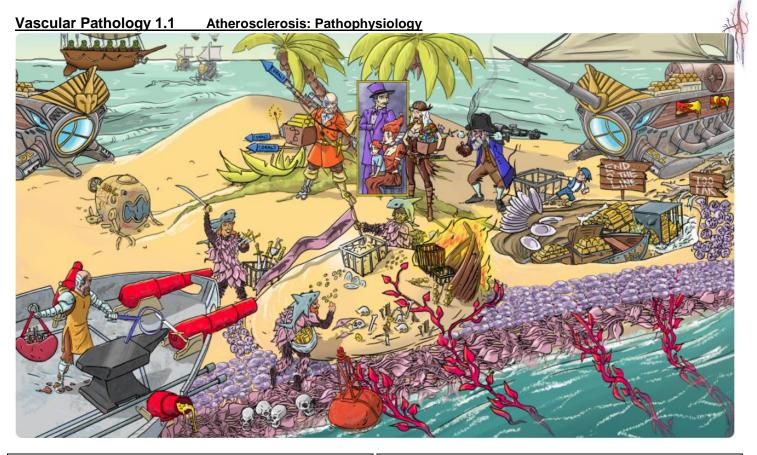
Type 4 RTA

- 33. Inspecting 4 "K" yellow test tubes : type 4 RTA
- 34. Crumbing MINERAL mountain ride: type 4 RTA is caused by DEFICIENCY of ALDOSTERONE
- 35. Abundant banana trees : type 4 RTA presents with HYPERKALEMIA (decreased aldosterone → potassium accumulation & sodium loss)
- 36. Fallen banana leading to loss of lemonade : in type 4 RTA, HYPERKALEMIA leads to INTRACELLULAR ALKALOSIS (excessive serum K+ \rightarrow K+ shifts INTO cells in exchange for H+ shift OUT of cells \rightarrow decreased H+ within cells
- 37. **Broken NH3 disposal buckets** : in type 4 RTA, intracellular alkalosis \rightarrow tubular cells DECREASE formation of AMMONIUM \rightarrow decreased secretion of H+ \rightarrow metabolic acidosis
- 38. **Getting rid of lemonade**: in type 4 RTA, a small amount of H+ is secreted into the tubule (due to mechanisms that do not require aldosterone)
- 39. Adding yellow lemonade to canoe water : in type 4 RTA, URINE pH is relatively NORMAL (acidic) (because some H+ is secreted into the tubule via non-aldosterone dependent mechanisms
- 40. **Destroyed RAIN umbrella**: type 4 RTA can be caused by LOW RENIN levels → decreased aldosterone synthesis (examples: diabetic nephropathy, NSAIDs cyclosporine → injure juxtaglomerular apparatus → low renin)
- 41. **Destroyed ACE card** : type 4 RTA can be caused by ACE INHIBITORS \rightarrow inhibit synthesis of angiotensin II \rightarrow decreased aldosterone synthesis
- 42. Heparin hunter : type 4 RTA can be caused HEPARIN ightarrow inhibition of zona glomerulosa of adrenal cortex ightarrow decreased aldosterone synthesis
- 43. **Torn adrenal beanie** : type 4 RTA can be caused by PRIMARY ADRENAL INSUFFICIENCY (Addison's disease) → decreased aldosterone synthesis)
- 44. **Proliferating adrenal hats** : type 4 RTA can be caused by CONGENITAL ADRENAL HYPERPLASIA (CAH) → decreased aldosterone synthesis)
- 45. $\mbox{\bf Spiral notebook}: \mbox{\bf SPIRONOLACTONE}$ can cause type 4 RTA (due to aldosterone RESISTANCE)
- 46. Almonds : AMILORIDE can cause type 4 RTA (due to aldosterone RESISTANCE)
- 47. **Tangerine**: TRIAMTERENE can cause type 4 RTA (due to aldosterone RESISTANCE)
- 48. **Stinky eggs**: SULFA DRUGS (trimethoprim) can cause type 4 RTA (by inhibiting aldosterone effects)



- 1. Red cannons obstructed by yellow gunk : atherosclerosis (accumulation of fatty plaques within blood vessels)
- 2. Inner sand dollar layer: TUNICA INTIMA (layer adjacent to vessel lumen) is composed of endothelium, connective tissue with basement membrane, and internal elastic lamina
- 3. **Smooth muscle seaweed**: SMOOTH MUSCLE of the TUNICA MEDIA (makes up the bulk of medium and large arteries)
- 4. Red seaweed infiltrating muscle seaweed layer: VASA VASORUM of the TUNICA EXTERNA (ADVENTITIA) (small vessels that penetrate and perfuse the outer $\frac{2}{3}$ of the tunica media)
- 5. Family portrait: a family history is the most important non-modifiable risk factor for atherosclerosis (usually multifactorial)
- 6. **Aging female pirate with "pause" helmet**: in women, menopause greatly increases the risk of atherosclerosis (the higher estrogen level in premenopausal women is protective)
- 7. Raising cholesterol chest: hypercholesterolemia is an important risk factor for atherosclerosis (circulates as esterified cholesterol within lipoproteins) (TOTAL cholesterol is sum of LDL, HDL, VLDL)
- 8. **Low-density pirate ship**: elevated low density lipoproteins (LDL) levels (lipoprotein with the highest cholesterol concentration) INCREASES risk of atherosclerosis ("bad" cholesterol)
- High-density submarine: elevated high-density lipoproteins (HDL) levels (transport cholesterol from peripheral tissues to the liver)
 DECREASES risk of atherosclerosis ("good" cholesterol)
- 10. **Trident on flying ship**: hypertriglyceridemia is a risk factor for atherosclerosis (often transported within very low density lipoproteins (VLDL)
- 11. **Raising candy chest**: diabetes is a risk factor for atherosclerosis (diabetes → dysfunctional lipid metabolism → increased LDL) (elevated insulin levels may also increase inflammation)]
- 12. Steaming angry : hypertension is a risk factor for atherosclerosis (endothelial injury \rightarrow formation of atherosclerotic plaque)
- 13. **Smoker**: smoking is a risk factor for atherosclerosis (causes endothelial injury)
- 14. **Raising HOMING beacon**: elevated HOMOcysteine levels are a risk factor for atherosclerosis (due to vascular injury)

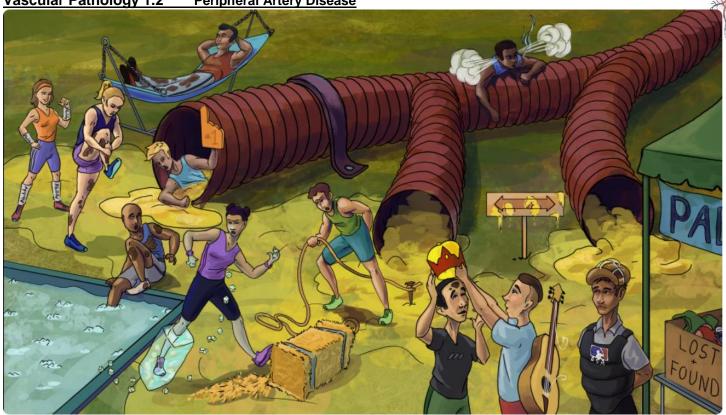
- 15. **Fallen foliage**: low folate levels are a risk factor for atherosclerosis (due to an increase in homocysteine levels)
- 16. **Discarded cobalamin cobalt fireworks**: low vitamin B12 levels are a risk factor for atherosclerosis (due to an increase in homocysteine levels)
- 17. **Cracked sand dollars**: ENDOTHELIAL INJURY is the initial step in formation of atherosclerotic lesions
- 18. **Stick piles**: endothelial injury creates a PROTHROMBOTIC STATE
- 19. "END O' THE LINe & Too Far" : endothelial injury leads to increased ENDOTHELIN and TISSUE FACTOR \rightarrow creates PROTHROMBOTIC STATE
- 20. Active LAD: endothelial injury leads to upregulation of LEUKOCYTE ADHESION MOLECULES (LAD) (includes ICAM, VCAM, selectin) \rightarrow promote leukocyte adhesion and activation at site of injury
- 21. **Dragging in MACROcage**: endothelial injury leads macroPHAGES to adhere and migrate into vessel wall
- 22. **PLATE activity**: endothelial injury (particularly exposure of subendothelial proteins) leads PLATELETS to aggregate and activate at the damaged site
- 23. **Burying chest-laden LOW DENSITY vessel** : endothelial injury leads LDL particles (containing cholesteryl esters) to migrate into the INTIMA



- 24. $\bf Sparks\ from\ MACROcage$: OXYGEN FREE RADICALS are formed by macrophages and local endothelial cells
- 25. Forming rust: LDLs become OXIDIZED as a result of exposure to oxygen free radicals
- 26. **Sea-foam engulfing cholesterol-laden MACROcage** : MACROphages phagocytose oxidized LDL → turn into "FOAM CELLS" (enlarged macrophages with abundant lipid in cytoplasm)
- 27. **Foam streaks**: FATTY STREAKS form as a result of FOAM CELLS accumulating beneath the endothelium (ubiquitous in people > 10 years of age)
- 28. CytoCOINS: FOAM cells release CYTOKINES and CHEMOKINES
- 29. **Squire-themed figurines**: T-CELLS migrate into the intima (as a result of chemotactic cytokines & increased expression of leukocyte adhesion molecules)
- 30. Platelets congregating with cytoCOINS : platelets continue to aggregate and activate \rightarrow release more cytokines and chemokines
- 31. Active fire: chronic INFLAMMATION is central to progression of atherosclerosis (inflammatory cells release cytokines \rightarrow attract more inflammatory cells \rightarrow release of more cytokines)
- 32. Bulging inner sand dollar layer : accumulation of inflammatory cells leads to INTIMAL THICKENING
- 33. Infiltrating "pink-seaweed pirates": SMOOTH MUSCLE CELLS (from the MEDIA) migrate into the INTIMA
- 34. **Seaweed pirates collecting cholesterol bars**: SMOOTH MUSCLE CELLS (altered by cytokines and growth factors) gain the ability to phagocytose LDL and cholesterol
- 35. Seaweed pirates wearing cartilage shark hats: altered SMOOTH MUSCLE CELLS synthesize excessive amounts of COLLAGEN and other extracellular matrix proteins
- 36. Cartilage shark flag over sand mound: SMOOTH MUSCLE CELLS proliferate and produce excessive COLLAGEN to form a FIBROUS CAPSULE over lipid core (creates an "ATHEROSCLEROTIC PLAQUE")
- 37. Bones, gold bars, and LDL ship debris buried in sand mound: the lipid core plaques contain areas of necrosis (with CALCIFICATION), cholesterol crystals, oxidized LDL, and cellular debris (foam cell and smooth muscle apoptosis)

- 38. **Infiltrating red seaweed**: plaques contain proliferations of thin-walled blood vessels (growth factors promote angiogenesis → vessel growth)
- 39. **Brandishing metal sword**: within plaques, macrophages and smooth muscle cells produce METALLOPROTEINASES → digest extracellular matrix (including fibrous cap)
- 40. **Skulls along pink seaweed**: atherosclerotic plaques can cause CYSTIC MEDIAL NECROSIS (severe thickening of intima and media → impaired perfusion of the MEDIA → necrosis of medial SMOOTH MUSCLE cells)
- 41. **Bulging red buoy**: atherosclerotic plaques can lead to ANEURYSM formation (often secondary to cystic medial necrosis)
- 42. Red cannon partially obstructed by yellow gunk: growth of atherosclerotic plaques can lead to vessel STENOSIS
- 43. "**70**" **tool design**: CRITICAL STENOSIS occurs if a plaque occludes more than 70% of the vessel lumen (may lead to onset of clinical manifestations)
- 44. **Anvil** : stenosis in coronary arteries (due to plaques) can cause angina
- 45. **Head with black paint-stroke**: stenosis in cerebral arteries (due to plaques) can cause transient ischemic attacks (TIA)
- 46. **Dirt clods**: stenosis in peripheral arteries (due to plaques) can cause claudication
- 47. **Kidney bag with narrow red straps**: plaques can cause renal artery stenosis

Vascular Pathology 1.2 Peripheral Artery Disease



- 1. **Yellow gunk in pipes**: peripheral artery disease (PAD)(atherosclerosis of non-cardiac vessels)
- 2. Proximal red pipe: abdominal aorta
- 3. Middle red pipe: common iliac artery
- 4. Outer red pipe: external iliac artery
- 5. **Black smoker**: black patients and smokers are at increased risk for PAD
- 6. Steaming ears: hypertension is a risk factor for PAD
- 7. **"#1" next to pipe beyond metal strap**: in PAD, lesions are most common in the superficial femoral artery (derives from the external iliac artery once it passes underneath the inguinal ligament)
- 8. **Diffuse yellow mud**: lesions in peripheral arteries are most commonly diffuse, bilateral, and affect multiple vessels
- 9. **Dirt clods**: PAD causes intermittent claudication (ischemic pain) (occurs in the distribution of the affected vessel)
- 10. Clutching leg while walking: claudication is produced by exertion
- 11. Resting happily: claudication resolves with rest
- 12. Clods on calf: calf claudication is caused by lesions in the superficial femoral artery
- 13. Clutching butt near iliac pipe: in PAD, buttock pain is caused by lesions in the internal iliac artery (or more distal arteries)
- 14. **Floppy rope near iliac pipe**: in PAD, erectile dysfunction is caused by lesions in the internal iliac artery (or more distal arteries)
- 15. Icy feet: PAD causes cool extremities (due to reduced blood flow)
- 16. **Baldness**: PAD causes hair loss in the affected extremity (due to deficient blood flow to hair follicles)
- 17. **Punched-out ankle**: PAD causes ischemic skin ulcers at pressure points (i.e lateral ankle)(deep lesions that appear "punched-out" with well-defined margins) (deficient blood flow → absence of bleeding or granulation tissue formation)
- 18. Acute ice leg: PAD can cause ACUTE LIMB ISCHEMIA
- 19. **Scattered embolic ice cubes**: in PAD, emboli can cause ACUTE LIMB ISCHEMIA (proximal atherothrombotic plaques break off and travel to smaller downstream arteries)
- 20. Ruptured thrombotic hay bail: in PAD, atheroma rupture and thrombosis in the affected artery can cause ACUTE LIMB ISCHEMIA

- 21. White painful leg stuck in ice: ACUTE LIMB ISCHEMIA causes pain, pallor, paralysis, poikilothermia (inability to regulate body temperature)
- 22. Watch at "0": ACUTE LIMB ISCHEMIA causes pulselessness
- 23. **Malfunctioning fitness tracker**: ACUTE LIMB ISCHEMIA causes paresthesias (tingling, numbness, burning)
- 24. **ABIlity ankle and wrist sweat bands**: PAD can be diagnosed using an ankle-brachial index (ABI) (SBP of ankles divided by SBP of upper extremities) (ABI is LOW in PAD)
- 25. **Red-yellow crown and broken guitar strings**: PAD is strongly associated with coronary artery disease (high risk of myocardial infarction)
- 26. **Stroke of mud on head**: PAD is strongly associated with cerebrovascular disease (high risk of stroke)
- 27. Umpire: aspirin is a treatment for PAD
- 28. Lost & found : cilostazol (a phosphodiesterase inhibitor) is a treatment for PAD)

Vascular Pathology 2.1 Hypertension

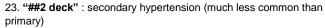


- 1. **Barometer** : <120/<80(normal); 120-129/<80(elevated); 130-139/80-89(stage 1); \geq 140/ \geq 90(stage 2)
- 2. "Primary" deck : primary hypertension (formally "essential htn) comprises >90#% of cases
- 3. Bucket volume: STROKE VOLUME x HR x SVR = MAP
- 4. Heart watch: SV x HEART RATE x SVR = MAP
- 5. Tight arterial rope: SV x HR x SYST VASC RESISTANCE = MAP
- 6. Carotid gauge : peripheral baroreceptors (carotid, aortic arch) \to modulate sympathetics (regulate HR, SVR, SV) \to SHORT TERM bp control
- 7. Rain umbrella : RENIN \rightarrow ATII (regulates SVR) \rightarrow aldosterone (regulates body vol. and therefore SV) \rightarrow SHORT TERM bp control
- 8. Chronic grandfather clock: LONG TERM bp control (pressure natriuresis)
- 9. Accumulating water and salty peanuts: impaired natriuresis (kidney dysfunction) is thought to contribute to the development of primary hypertension (increased Na+ reabsorption, decreased Na+ excretion)
- 10. **High pressure washer** : impaired natriuresis (kidney dysfunction) → blood pressure rises until salt/water excretion returns to normal (PRESSURE natriuresis) → chronic primary hypertension
- 11. **Fight or flight with peanuts**: chronic sympathetic activity is thought to contribute to the development of primary hypertension (increases Na+resorption at nephron → blood pressure rises until salt/water excretion returns to normal (PRESSURE natriuresis))
- 12. $\mbox{\bf Aged Ahab}$: age is a risk factor for development of primary hypertension

- 13. **Old isolated spraying prisoner**: hypertension in elderly commonly presents as ISOLATED SYSTOLIC hypertension
- 14. **Refuse compliance**: DECREASED COMPLIANCE of large arteries → ISOLATED SYSTOLIC hypertension in elderly
- 15. **Shark tattoo + calcium deposits** : accumulation of collagen and calcium in artery wall→ decreased compliance → ISOLATED SYSTOLIC hypertension in elderly
- 16. **Degraded shorts** : degraded elastin in large arteries \rightarrow decreased compliance \rightarrow ISOLATED SYSTOLIC hypertension in elderly
- 17. **Increased distance between bars**: hypertension in elderly is often associated with increased PULSE PRESSURE (increased systolic, decreased diastolic)
- 18. **Older black male**: black patients are at increased risk of developing primary hypertension (presents sooner, more severe)
- 19. **Salty peanuts**: high sodium intake (risk factor for development of primary hypertension)
- 20. **EtOH jug**: excessive alcohol intake (risk factor for development of primary hypertension)
- 21. **Rotund and sitting**: obesity and sedentary lifestyle (risk factors for development of primary hypertension)]
- 22. **Family photo**: family history (risk factor for development of primary hypertension)

Vascular Pathology 2.1 Hypertension

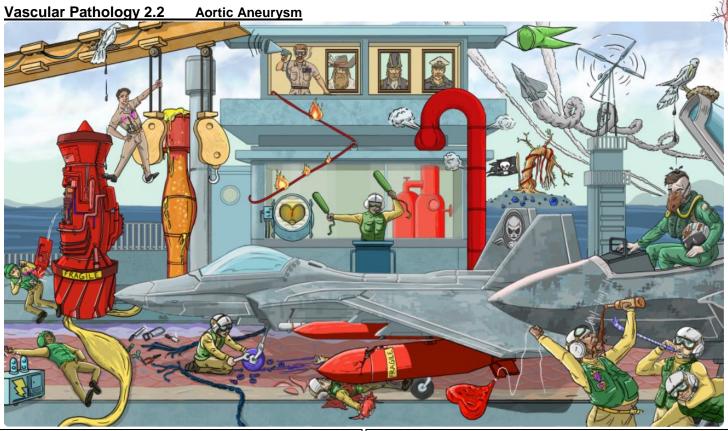




- 24. **Stenosed kidney rigging**: renovascular hypertension (stenosis of renal artery → reduced renal perfusion → increased RAAS) (2*hypertension)
- 25. Rain umbrella: stenosis of renal artery \rightarrow reduced renal perfusion \rightarrow increased RENIN from JGA \rightarrow ATII (vascoconstriction) and aldosterone (vol retention) activity (renovascular hypertension)
- 26. **Gunky renal rigging**: renovascular hypertension is usually caused by ATHEROSCLEROSIS of renal artery (reduced renal perfusion → increased RAAS)
- 27. **Muscular lady with collagen shark tattoo**: fibromuscular dysplasia more common in women (collagen deposition/smooth muscle hyperplasia in renal artery → reduced renal perfusion → increased RAAS) (2*hypertension)
- 28. **Bead strings**: "string of beads" appearance of renal artery characteristic of fibromuscular dysplasia (fibromuscular hyperplasia alternating with regions of aneurysm)
- 29. **Neck beads**: many patients with fibromuscular dysplasia also have carotid involvement (headache, tinnitus, increased risk of TIA, stroke)
- 30. **Sound waves**: auscultate for bruits (turbulent flow) in renal arteries and carotids (renovascular hypertension/fibromuscular dysplasia)
- 31. **Small kidney pulley**: poorly perfused kidney atrophies → asymmetric kidney sizes (renovascular hypertension/fibromuscular dysplasia)
- 32. **Discarded aces**: adverse reaction to ACE therapy (patients with renovascular hypertension/fibromuscular dysplasia rely on ATII to constrict efferent arterioles \rightarrow preserve GFR)
- 33. 2 loose red suspenders and spilled GFR coffee : ACE inhibitor \rightarrow decreased ATII activity \rightarrow decreased GFR (patients with renovascular hypertension/fibromuscular dysplasia rely on ATII to constrict efferent arterioles \rightarrow preserve GFR)
- 34. **Broken kidney pulleys**: acute and chronic kidney disease can cause 2° hypertension (increased renin and/or decreased salt and water excretion)
- 35. **Mortar and pestle**: several classes of drugs can cause 2° hypertension (OCPs, NSAIDs, corticosteroids, cocaine/meth)
- 36. **Mineral crystals** : hyperaldosteronism (mineralocorticoid) \to salt/water conservation \to 2° hypertension and hypokalemia
- 37. Snoring sailor : obstructive sleep apnea is associated with 2° hypertension

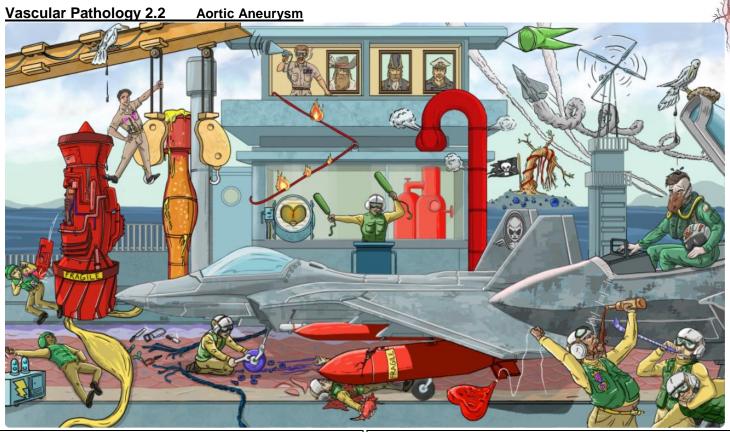
- 38. Concentric conch and floppy balloon : chronic hypertension \to concentric hypertrophy (increased afterload on heart) \to heart failure
- 39. **Dissected pipe**: chronic hypertension is associated with aortic dissection
- 40. Gunky pipe : chronic hypertension \rightarrow endothelial damage \rightarrow atherosclerosis
- 41. Dilated buoy : chronic hypertension \to vessel wall damage \to increased risk of aortic aneurysm)
- 42. Broken string and tattooed head: chronic hypertension \rightarrow atherosclerosis/arteriosclerosis \rightarrow increased risk of CVA and ischemic stroke
- 43. **Bloody head** : chronic hypertension \rightarrow arteriosclerosis \rightarrow increased risk of hemorrhagic stroke
- 44. Retinal life preserver : chronic hypertension \rightarrow retinopathy (retinal hemorrhages/exudates) and papilledema
- 45. **Kidney stuck in whirlpool** : chronic hypertension → nephrosclerosis/glomerulosclerosis → CKD → worsens hypertension...
- 46. **180 protractor/120 ruler** : hypertensive URGENCY (>180/120 WITHOUT end organ damage)
- 47. **180 protractor/120 ruler and damaged ship**: hypertensive EMERGENCY (>180/120 PLUS end organ damage: encephalopathy, ICH, papilledema, AKI, pulmonary edema, or HF)
- 48. **brain hat, life preserver, cracked kidney, floppy balloon**: common sx of hypertensive EMERGENCY include encephalopathy, ICH, papilledema, AKI, pulmonary edema, or HF)
- 49. **Scaly sclerotic snake**: chronic hypertension → hyaline arterioloSCLEROSIS (hyaline deposition in vessel wall)
- 50. **Leaky hull** : chronic hypertension → endothelial damage → LEAK of plasma proteins into media (hyaline arteriolosclerosis)
- 51. **Pink algae**: on histology, vessel walls thickened due to hyaline deposition (hyaline arteriolosclerosis)
- 52. **Tiny portholes**: luminal narrowing (vessel walls thickened due to hyaline deposition) (hyaline arteriolosclerosis)
- 53. **Glomerular rope**: chronic hypertension → glomerulosclerosis (hyaline deposition/ damage of glomerular capillaries)
- 54. **Onions**: chronic severe hypertension/hypertensive emergency → hyperplastic arteriolosclerosis (smooth muscle hypertrophy, basement membrane replication, concentric thickening) → "onion skin" appearance)
- 55. **Fibrous gunky dreadlock beanie with skull beads**: hypertensive emergency → fibrinoid necrosis of vessel wall (accumulation of necrotic debris and fibrin (pink) in vessel wall, inflammatory infiltrate, luminal narrowing/obliteration)





- 1. **Purple, red, and tan runway**: TRUE aortic aneurysms involve ALL layers of the vessel wall (intima, media, adventitia), as opposed to FALSE aneurysms (only involve the adventitia)
- 2. **Bulging under kidney crane**: most AAA form BELOW the renal arteries (involvement of the renal arteries can cause renal injury and even failure)
- 3. Yellow gunk in pipe: many patients with AAA also have atherosclerosis, even right inside the aneurysm itself (it is thought that these two processes share the same risk factors, though it's unclear if atherosclerosis directly causes AAA)
- 4. **Seasoned commander**: age (especially >60 years) is an important risk factor for aortic aneurysm (as well as thoracic aneurysm)
- 5. **Smoker**: smoking is a risk factor for aortic aneurysm (positive correlation between smoking history and aneurysm size
- 6. **Bulge in ascending red pipe**: the aortic arch is the second most common location of aortic aneurysm (many patients also have AAA)
- 7. **Steaming pipe**: thoracic aortic aneurysm has many of the same risk factors as atherosclerosis, especially HYPERTENSION (present in 60% of cases)
- 8. **Marfan martian**: Marfan syndrome (mutation of fibrillin in elastic tissue) decreases distensibility of the aorta and can lead to aortic aneurysm formation (especially in the thoracic aorta)
- Bicuspid windsock : bicuspid aortic valves (in which there are only two leaflets) can cause aortic root dilation→ thoracic aortic aneurysm formation
- 10. Wind marker turning : Turner syndrome (XO) is associated with bicuspid aortic valves \rightarrow dilation of the aortic root \rightarrow thoracic aortic aneurysm formation
- 11. Family portraits: family history is a risk factor for aortic aneurysm
- 12. **Blue nuts and bolts**: inflammatory cell infiltrate in vessel wall (seen in cystic medial degeneration)
- 13. **Snapped elastic wire**: degradation of elastic lamina (seen in cystic medial degeneration)
- 14. **Sharp metal tools**: degradation of extracellular matrix by metalloproteinases (seen in cystic medial degeneration)
- 15. **Broken smooth muscle tiles with blue gunk**: smooth muscle apoptosis and necrosis→ accumulation of mucopolysaccharide (seen in cystic medial degeneration)

- 16. **Vibrating walkie talkie on belt**: AAA are often found on physical exam as a palpable, pulsatile abdominal mass
- 17. **Ultrasound bullhorn**: screening abdominal ultrasound should be performed on men >65 with a history of smoking (AAA)
- 18. **Bird poop on shoe**: distal embolization of thrombus within aortic aneurysms can cause acute limb ischemia
- 19. **Bulging yellow hose**: AAA large enough can obstruct the ureters→ hydronephrosis and postrenal kidney injury
- 20. **"FRAGILE" across pipe diameter**: risk of aortic aneurysm rupture increases with vessel diameter (increased vessel radius→ increased wall tension and hemodynamic stress)
- 21. Ruptured piece hitting flank: retroperitoneal hemorrhage in AAA rupture— severe, acute flank pain
- 22. **Pulsing walkie talkie being hit**: severe flank pain and a pulsatile abdominal mass are suggestive of ruptured AAA
- 23. **Lightning bolt**: severe hemorrhage in aortic aneurysm rupture→ hypovolemic shock
- 24. Fallen sailor by lightning fuse: severe hemorrhage in ruptured AAA is associated with a 50% mortality rate



- 25. **Party blower**: thoracic aortic aneurysms large enough can cause tracheal compression→ cough, wheezing, or dyspnea
- 26. **Call sign "horse"** : thoracic aortic aneurysms can compress the left recurrent laryngeal nerve (due to it location in the aortic arch) \rightarrow hoarseness
- 27. **Choked in headlock**: thoracic aortic aneurysms large enough can compress the esophagus→ dysphagia
- 28. Expanded waistline : aortic arch aneurysms extending proximally can cause dilation of the aortic root → aortic regurgitation
- 29. **Regurging liquid**: there is a characteristic diastolic decrescendo murmur in aortic regurgitation
- 30. **Floppy heart balloon**: untreated or severe acute aortic regurgitation can cause heart failure (TAA)
- 31. **Purple heart crown**: thoracic aortic aneurysms may extend proximally and block the coronary artery ostia→ ischemic heart disease
- 32. **Bird poop on head**: embolism of thrombus within thoracic aneurysms can travel through the carotids into cerebral circulation→ stroke
- 33. "FRAGILE" chest missile: risk of rupture of thoracic aortic aneurysms increases with increasing vessel diameter (especially >6cm)
- 34. **Chest-crushing missile**: ruptured thoracic aortic aneurysm presents with acute, tearing chest pain radiating to the back
- 35. **Passed out under missile**: severe hemorrhage in thoracic aortic aneurysm rupture can lead to hypovolemic shock
- 36. **Gripping heart**: thoracic aneurysm rupture into the pericardium can lead to cardiac tamponade
- 37. **Bacterial signal lanterns**: infection of injured portions of the aorta (atherosclerotic plaque, catheterization injury, pre-existing aneurysm) can cause/worsen aortic aneurysm (mycotic aneurysm)
- 39. **Red hose on fire**: inflammatory vasculitides affecting the aorta (temporal arteritis, Takayasu arteritis) can lead to aortic aneurysm formation

- 40. **Spiral jet smoke**: tertiary syphilis (caused by the spirochete T. pallidum) can cause aortitis and aortic aneurysm formation (especially thoracic)
- 41. **Red vines on tree**: syphilitic aortitis begins with destruction of the vasa vasorum (which perfuses the outer ¾ or the aortic wall) in the aortic arch→ cystic medial degeneration→ aneurysm formation
- 42. **Fibrous tree**: extensive fibrosis of the aorta in syphilitic aortitis gives it a "tree bark" appearance
- 43. **Blue shells around tree**: syphilitic aortitis causes an intense, plasma cell rich, inflammatory infiltrate
- 44. **Pirate CXR flag with linear contrail**: syphilitic aortitis causes linear calcification of the aortic arch, which can be seen on CXR

Vascular Pathology 2.3 Aortic Dissection



- Fluid between hatch and smooth muscle tile: in aortic dissection, tears in the tunica intima allow blood to expand into the tunica MEDIA (the smooth muscle layer of vessels)
- 2. **Steam from ears**: chronic hypertension is the most important risk factor for aortic dissection (though acute elevations in blood pressure, as with cocaine or amphetamine use, can cause dissection as well)
- 3. **Decorated captain**: most aortic dissections occur in older males (typically over age 60)
- 4. Loose screws: Ehlers-Danlos syndrome (disorders of collagen production) predispose younger patients to aortic dissection and even rupture
- 5. **3 gilled shark** : Type III collagen is an important structural component of blood vessel walls and is mutated in Ehlers-Danlos syndrome→ increased risk of aortic dissection or rupture
- 6. **Martian**: Marfan syndrome (a mutation in fibrillin → defective elastic tissue) causes dilation of the aorta and aortic root, predisposing younger patients to aortic dissection
- 7. **Pipe cleaning tool** : damage to the intimal layer resulting in aortic dissection can be iatrogenic (as with cardiac catheterization)
- 8. Aneurysmal pipe: aortic aneurysms are highly associated with intimal damage and vessel wall weakening— aortic dissection
- Swelling around pipe lumen: in acute aortic dissection, blood expanding into the smooth muscle media initially causes dilation of the vessel wall (resembling an aneurysm)
- 10. **Hollow pipe**: separation of the medial smooth muscle layer by dissecting blood creates true (systemic bloodflow) and false (dissecting bloodflow) lumen
- 11. Degenerating smooth muscle tiles: cystic medial degeneration (a process that leads to mucoid degeneration of the medial smooth muscle layer) weakens vessel walls \rightarrow aortic dissection
- 12. **Arching pipe**: aortic dissection most commonly occurs in portions of the aortic arch (though any vessel is prone to dissection)
- 13. **High pressure bend**: up to 30% of thoracic aortic dissections occur at the takeoff of the left subclavian artery (arterial branch points endure high hemodynamic stress)
- 14. **Area A**: thoracic aortic dissections involving the ASCENDING aorta are classified as type A in the Stanford system (dissections that spread to the descending aorta are still classified as type A because of the involvement of the ascending aorta)
- 15. Area B: thoracic aortic dissections involving the DESCENDING aorta ONLY are classified as type B in the Stanford system
- 16. **Zone I**: thoracic aortic aneurysms involving BOTH the ascending and descending aorta are classified as type I in the DeBakey system
- 17. Zone II: thoracic aortic aneurysms involving the ASCENDING aorta ONLY are classified as type I in the DeBakey system
- 18. **Zone III**: thoracic aortic aneurysms involving the DESCENDING aorta ONLY (defined as any area distal the the left subclavian artery) are classified as type III in the DeBakey system

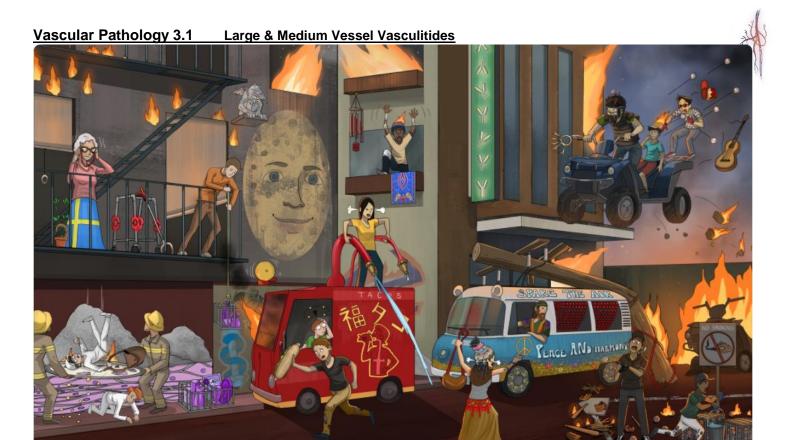
- 19. **Back of shirt tearing**: descending thoracic aortic dissections (type B or III) often present with tearing BACK or POSTERIOR chest pain (most commonly between the scapulae)
- 20. Front of shirt tearing : ascending thoracic aortic dissections (type I, II, or A) often present with tearing ANTERIOR chest pain radiation to the back
- 21. **SINKING**: occlusion of the carotid arteries by an expanding thoracic aortic dissection can reduce cerebral blood flow, causing syncope
- 22. **Black stroke on head**: occlusion of the carotid arteries by an expanding thoracic aortic dissection can reduce cerebral blood flow→ ischemia→ ischemic stroke
- 23. Low pressure gauge : aortic dissections can expand all the way to the aortic bifurcation and occlude the iliac arteries→ pulselessness and ischemia in the lower extremities
- 24. **System malfunction**: aortic dissections can expand and occlude ANY vessel branching from the aorta, resulting in end organ dysfunction (such as renal failure, mesenteric ischemia, and even spinal cord ischemia)
- 25. **Broken heart string** : ascending aortic dissections can block the coronary ostia (which arise from the proximal ascending aorta) \rightarrow myocardial ischemia \rightarrow myocardial infarction
- 26. **Regurgent sailor**: ascending aortic dissections can expand proximally and involve the aortic root→ dilation of the aortic annulus → acute aortic regurgitation
- 27. **Murmur lines**: acute aortic regurgitation results in a new onset decrescendo diastolic murmur
- 28. Floppy heart balloon: acute aortic insufficiency in aortic dissection can lead to acute heart failure and cardiogenic shock
- 29. **Squeezing heart** : ascending aortic dissection may rupture into the pericardium, allowing blood to fill the pericardial space→ cardiac tamponade
- 30. **Lightning bolt**: thoracic aortic dissection often presents with signs and symptoms of cardiogenic shock (secondary to acute MI, severe aortic regurg, or cardiac tamponade)
- 31. WARNING VOLUME: in aortic dissection, enough blood can leave systemic circulation (filling the false lumen) to cause hypotension
- 32. **Lightning bolt**: in aortic dissection, large amounts of blood can be diverted from systemic circulation to the false lumen through the intimal tear→ hypovolemic shock
- 33. **Burst pipe** : aortic dissection can lead to progressive vessel wall weakening \rightarrow aortic rupture \rightarrow hypovolemic shock and even death
- 34. Extra wide tie by pirate flag : aortic dissection causes dilation of the aorta→ widened mediastinum on chest xray
- 35. **CAT scan cat**: in aortic dissection, CT angiography will show a widened aorta with a "double lumen" (true and false lumen) separated by a dark band of tissue (flap of dissected intimal tissue)
- 36. **Pressure valve**: in aortic dissection, strict blood pressure control is required to decrease the spread of dissection

Vascular Pathology Large & Medium Vessel Vasculitides



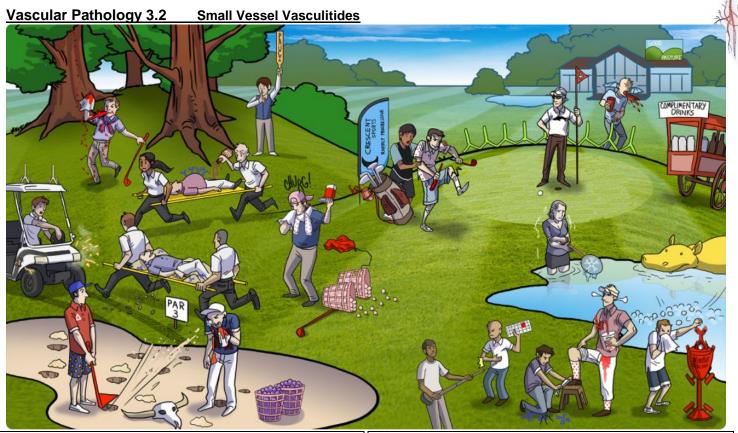
- 1. Hat on fire: recurrent fever is common in systemic inflammatory diseases
- 2. Really thin: weight loss is common in systemic inflammatory diseases
- 3. Wrapped joints: arthralgias are common in systemic inflammatory diseases
- 4. Elevated cylindrical wind chime: erythrocyte sedimentation rate (ESR) measures plasma viscosity and is elevated in inflammatory conditions such as systemic vasculitis
- 5. **High CaRPet**: C-reactive protein (CRP) is an acute phase reactant elevated in inflammatory conditions such as systemic vasculitis
- 6. Arterial glasses strap: giant cell (temporal) arteritis preferentially affects cranial branches of the carotid artery (especially the temporal artery)
- 7. Arched branching plant: giant cell arteritis affects the proximal aorta, carotid arteries, and carotid branches (especially the OPHTHALMIC artery)
- 8. Old lady on burning balcony: giant cell arteritis most commonly affects women over 50 (peak incidence around 70)
- 9. Scandinavian skirt: there is a markedly increased incidence of giant cell arteritis in people of Scandinavian descent
- 10. Myalgic jointed walker: up to half of patients with giant cell arteritis also have polymyalgia rheumatica (rheumatic arthralgia and stiffness of the shoulders, hips, neck, and torso)
- 11. Outer pavement : tunica adventitia
- 12. Smooth muscle tile: smooth muscle tunica media
- 13. Elastic trampoline: internal elastic lamina
- 14. Inner ash pile: endothelium
- 15. Helper kids in white on trampoline and smooth muscle tiles histologically, giant cells arteritis shows CD4+ helper T cell inflammation of the media and internal elastic lamina
- 16. MacroCAGES full of purple objects: in giant cell arteritis, macrophages accumulate in the media and form multinucleated giant cells
- 17. Patchy flames: inflammation in giant cell arteritis is patchy and segmental
- 18. Holes in elastic trampoline: fragmentation of the internal elastic lamina due to inflammation and medial fibrosis is seen in giant cell arteritis
- 19. Thick ash pile: inflammation in giant cell arteritis causes intimal thickening and nodule formation \rightarrow luminal narrowing \rightarrow distal ischemia
- 20. Gargoyle claw to jaw: jaw claudication (pain with chewing or talking) is common in giant cell arteritis
- 21. Wincing headache: headache and tenderness to palpation over the temporal areas are common in giant cell arteritis
- 22. Foggy glasses: inflammation of the ophthalmic artery in giant cell arteritis can lead to visual changes ranging from decreased acuity to complete vision loss

- 23. Window shade pulled down: amaurosis fugax, a transient "descending curtain" pattern of vision loss, is seen in giant cell arteritis due to episodic occlusion of the central retinal or posterior ciliary arteries of the eye
- 24. Broken window shade: inflammation in giant cell arteritis can lead to PERMANENT vision loss (patients require annual fundoscopic exam)
- 25. Extended grabber: diagnosis of giant cell arteritis is confirmed with histologic exam of a temporal artery biopsy
- 26. Eerie moon face: treatment of giant cell arteritis is immunosuppression with corticosteroids
- 27. Asian woman fighting fire: Takayasu arteritis most commonly affects women of Asian descent under 40
- 28. Arched taco: Takayasu arteritis has a predilection for the aortic arch and its proximal branches (though inflammation can affect abdominal and cerebral vessels)
- 29. Spicy tacos in tummy: involvement of the abdominal aorta and renal arteries occurs in up to half of cases of Takayasu arteritis
- 30. Ash wall: Takayasu arteritis exhibits similar histology to GCA (T-cell infiltrate of media/elastic lamina, granulomas, intimal thickening)
- 31. Different pressure in hoses: inflammation and narrowing of proximal aortic vessels can lead to significantly different blood pressures (and even absent pulses) in the extremities in both giant cell and Takayasu arteritis
- 32. Fire alarm ringing: inflammation and luminal narrowing of vessels can lead to turbulent blood flow and audible bruits (especially of the carotid and abdominal aortic arteries)(GCA + Takayasu)
- 33. Bulge in hose: inflammation in both giant cell and Takayasu arteritis can lead to aneurysm formation and aortic root dilation→ aortic regurgitation
- 34. Steaming ears: luminal narrowing of the renal arteries in Takayasu arteritis→ renal hypoperfusion→ severe renovascular hypertension
- 35. Seeing stars through foggy glasses: inflammation of the carotid and cerebral arteries can lead to neurologic symptoms such as vertigo, syncope, and visual changes
- 36. Stealing sub: inflammation and stenosis of the subclavian artery PROXIMAL to the vertebral artery takeoff can cause RETROGRADE flow through the vertebral artery→ vertigo, visual changes, syncope (subclavian steal syndrome)
- 37. Dirt clods on legs: inflammation of the distal aorta and proximal iliac vessels → vessel narrowing → limb claudication (pain with walking relieved with rest)]



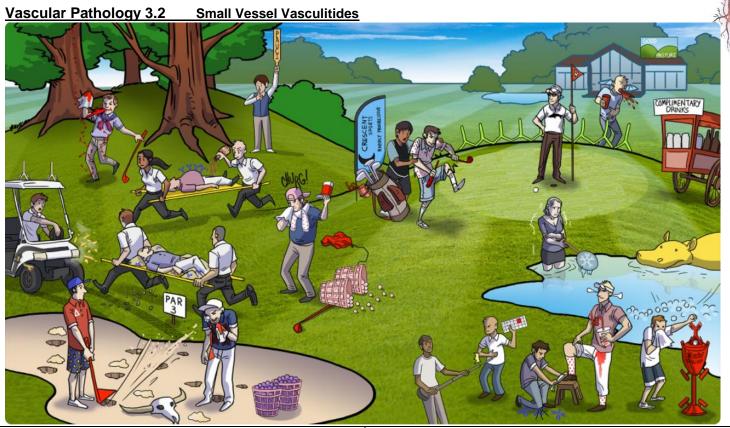
- 38. **Peace ANd harmony**: polyarteritis nodosa (PAN) is a diffuse vasculitis of medium and small vessels involving multiple organs
- 39. **Scattered flowers**: PAN causes acute flares and relapses with a background of chronic inflammation, leading to lesions of varying age and severity
- 40. **Hippo hippie van**: PAN has a strong association with hepatitis C and especially hepatitis B infection
- 41. **3 prong peace sign antibody wheels**: PAN is caused by immune complex deposition in tissues→ complement activation, inflammation, and tissue damage (type III hypersensitivity reaction)
- 42. **String of beads**: histologically, PAN is marked by SEGMENTAL, TRANSMURAL NECROTIZING inflammation→ microaneurysm formation and alternating areas of stricture and dilation ("string of beads" vessel appearance)
- 43. **Blue dots on beanie**: PAN features a NON-granulomatous mixed inflammatory infiltrate of the ENTIRE vessel wall
- 44. **Pink gunk and skulls in dreadlocks**: inflammation in PAN leads to fibrinoid necrosis in vessels (accumulation of necrotic cells, eosinophilic fibrin, and immune complexes)
- 45. **Steam from hippie ears**: renal involvement in PAN commonly causes hypertension
- 46. **Kidney purse with red straps**: renal vessel involvement in PAN can lead to glomerulonephritis, renal infarction, AKI, and chronic kidney disease
- 47. **Henna tattoo**: up to a third of patients with PAN will develop skin changes including subcutaneous nodules, palpable purpura, and livedo reticularis (a purplish reticular discoloration of skin)
- 48. **Broken telephone pole**: peripheral nerve involvement in PAN can lead to motor and, less commonly, sensory loss
- 49. **Red belly macrame**: PAN may cause mesenteric arteritis, leading to abdominal pain and, if severe, chronic mesenteric ischemia
- 50. "Spare the air" : PAN often spares the pulmonary vasculature $\,$
- 51. Young Asian boy on ATV: Kawasaki disease is a systemic vasculitis most commonly seen in young males of Asian descent
- 52. **CRASHED Kawasaki ATV**: features of Kawasaki disease include Conjunctival injection, desquamative Rash, Adenopathy (cervical), Strawberry tongue, and erythema of Hands and feet
- 53. **Burning hat**: Kawasaki disease usually causes high-spiking fever lasting at least FIVE DAYS

- 54. Red goggles: conjunctival injection is common in Kawasaki disease
- 55. **Red spotted shirt**: desquamative rash of the trunk is common in Kawasaki disease
- 56. **Ball near neck**: cervical adenopathy (nodes >1.5cm) is common in Kawasaki disease
- 57. **Sticking tongue out**: "strawberry tongue" (with inflammation of the lips and mouth as well) is common in Kawasaki disease
- 58. **Red gloves and cleats** : erythema and swelling of the hands and feet are common in Kawasaki disease
- 59. **Dilated red crown falling off**: inflammation of the coronary vessels in Kawasaki disease can lead to coronary artery aneurysms
- 60. **Broken heart string**: coronary artery aneurysm development in Kawasaki disease predisposes→ thrombosis and MI (the most common cause of death in Kawasaki disease)
- 61. **ASA umpire dropping Ig keys**: treatment of Kawasaki disease consists of a combination of IVIG and high-dose aspirin (the ONLY pediatric infectious disease in which aspirin is used (risk of Reye's syndrome))
- 62. **Smoking cigar and calling TO**: thromboangiitis obliterans (TO) or Buerger disease is STRONGLY associated with tobacco smoke
- 63. Burning sticks and home plate : the key finding in TO is THROMBUS formation that leads to OBLITERATION of vessel lumen \rightarrow distal ischemia
- 64. **Balls in macroCAGES**: TO involves a GRANULOMATOUS lymphocytic infiltrate with microabscess and thrombus formation→ obliteration of vessel lumen
- 65. Burning telephone pole behind guy smoking: spread of inflammation to surrounding structures in thromboangiitis obliterans can cause superficial phlebitis of the veins or nerve inflammation→ neuropathy and pain
- 66. **Fingerless gloves with blue fingers**: patients with thromboangiitis obliterans often suffer Raynaud phenomenon
- 67. **Dirt clods on baseball pants**: patients with thromboangiitis obliterans often have intermittent claudication (pain with activity relieved with rest) due to decreased blood flow
- $68. \, \textbf{Black gloves}$ and cleat tips : inflammation in TO can lead to ulcers and even gangrene of the fingers and toes
- 69. **Hives on no smoking sign**: many patients with TO will demonstrate a hypersensitivity reaction to dermal injection of tobacco extract



- 1. **Grabbing red spots**: a common feature of small vessel vasculitis (MPA, GPA, Churg-Strauss, IgA nephropathy, Good pasture) is PALPABLE purpura (raised, non-blanching, diffuse erythematous papules and plaques)
- 2. **Bendy red glomerular straw**: glomerulonephritis is a common feature of small vessel vasculitis (MPA, GPA, Churg-Strauss, IgA nephropathy, Good pasture) ranging from mild hematuria & proteinuria to AKI and acute renal failure
- 3. **Crescent logo**: many small vessel vasculitis syndromes (MPA, GPA, Churg-Strauss, IgA nephropathy, Good pasture) are associated with rapidly progressive glomerulonephritis (RPGN) characterized by crescent formation within glomerulus
- 4. **Lung pockets**: many small vessel vasculitis syndromes (MPA, GPA, Churg-Strauss, IgA nephropathy, Good pasture) are associated with PNEUMONITIS, which can present as infiltrates, asthma, interstitial fibrosis, and alveolar hemorrhage.
- 5. First responder stretcher with antibody SAND INSIDE: c-ANCA (anti-neutrophilic cytoplasmic antibody), or anti-proteinase-3, antibodies are directed against proteinase-3 in CYTOPLASMIC neutrophil GRANULES.
- 6. PAR 3 : c-ANCA autoantibodies are directed against CYTOPLASMIC proteinase-3 (PR3-ANCA)
- 7. First responder stretcher with peri-abdominal antibodies: p-ANCA (anti-neutrophilic cytoplasmic antibody), or anti-myeloperoxidase, antibodies are directed against PERINUCLEAR myeloperixodase
- 8. **EMT pouring MPO**: p-ANCA autoantibodies are directed against PERINUCLEAR myeloperoxidase (MPO)
- 9. **Antibodies on roof of golf cart** : sensitized neutrophils of individuals with a predisposition to vasculitis development start to express proteinase-3 and myeloperoxidase on cell SURFACES→ autoantibody binding and neutrophil activation
- 10. Cyto-coins and sparks from overexcited first responders : activation of neutrophils by ANCA antibodies → production of cytokines/reactive oxygen species → inflammation/endothelial damage
- 11. **Damaging delicate inner lawn**: cytokine release and inflammation caused by ANCA-activated neutrophils causes endothelial damage→ vasculitis

- 12. **Sand WEDGE next to cytoplasmic EMTs**: Wegener's granulomatosis (granulomatosis with polyangiitis, GPA) is a c-ANCA positive disease most commonly seen in older men
- 13. **Breathing in sand spray**: development of c-ANCA antibodies in GPA most likely follows airborne environmental antigen exposure in predisposed individuals
- 14. **Golf ball macro-cages**: GPA causes a necrotizing vasculitis with granuloma formation (multinucleated giant cells on histology)
- 15. Necrotic bull skull: granulomas in GPA often have central necrosis
- 16. **Triangle logo**: GPA presents with the triad of upper respiratory symptoms, pulmonary symptoms, and glomerulonephritis
- 17. **Bloody tissue** : upper respiratory involvement in Wegener's granulomatosis usually presents as chronic rhinosinusitis→ congestion and purulent nasal discharge
- 18. **Bloody nose from ball strike**: necrotizing granulomatous inflammation in Wegener's causes mucosal ulceration and tissue destruction (such as septal perforation→ "saddle nose" deformity")
- 19. **Divots and clumps in Wegener bunker**: pulmonary involvement in Wegener's→ recurrent pneumonia, interstitial fibrosis, cavitary lesions, and hemorrhagic nodules
- 20. **Tiny ants next to peri-abdominal first responders**: microscopic polyangiitis (MPA) is a p-ANCA associated diffuse vasculitis of small vessels (arterioles, venules, and capillaries)
- 21. **Red lung scarf on chest**: microscopic polyangiitis has a predilection for pulmonary vessels→ recurrent pneumonia, pneumonitis, interstitial fibrosis
- 22. **Spitting bloody ants** : pulmonary vessel damage in microscopic polyangiitis can cause alveolar hemorrhage \to dyspnea and hemoptysis
- 23. Pauci on the ANCA greens: glomerulonephritis is common to the ANCA-positive vasculitides (GPA, MPA, Churg-Strauss) but does NOT cause immune complex deposition in glomeruli ("pauci" immune on microscopy)



- 24. **Spilling pink balls next to peri-abdominal first responders**: Churg-Strauss syndrome (EOSINOPHILIC granulomatosis with polyangiitis) is a p-ANCA vasculitis associated with eosinophilic tissue infiltrates and peripheral eosinophilia
- 25. **CHURG! sneeze**: Churg-Strauss syndrome (EOSINOPHILIC granulomatosis with polyangiitis) is likely due to exposure to an unknown allergen, infection, or drug→ atopy and development of p-ANCA antibodies
- 26. Churg-rash : atopy is common in Churg-Strauss \to eczema, allergic rhinitis, nasal polyps, and atopic dermatitis
- 27. **Inhaler**: almost all patients with Churg-Strauss syndrome have asthma (glucocorticoid treatment for respiratory symptoms often masks vasculitis for years)
- 28. **Pink dot sleeve on chest**: eosinophilic infiltration of the lungs in Churg-Strauss→ pulmonary infiltrates, nodules, and effusion
- 29. **Pink ball macro-cages**: Churg-Strauss syndrome causes GRANULOMATOUS inflammation with eosinophilia, tissue necrosis, and formation of multinucleated giant cells
- 30. **Floppy heart balloon by pink balls**: eosinophilic myocarditis in Churg-Strauss can lead to fibrosis and congestive heart failure (up to 50% of patients have myocardial involvement)
- 31. **Frayed ANCA cart wiring**: inflammation in ANCA-associated vasculitis may spread to surrounding nerves, causing pain, paresthesias, or paresis (mononeuritis multiplex is the term for damage to at least two nerves in two different locations)
- 32. **Henoch shoe shine**: Henoch-Schonlein purpura (HSP) is a type III hypersensitivity reaction (immune complex deposition) causing vasculitis in young children
- 33. **Sneezy young caddy**: most cases of HSP follow an upper respiratory infection (flu or S. pneumo)
- 34. Complex IgA ball washer: HSP is thought to be due to defective mucosal immunity→ IgA immune complex deposition in tissue
- 35. "Complimentary ball wash": deposition of IgA immune complexes in tissue causes complement activation and inflammation (C3 deposits are seen alongside IgA on immunohistochemistry)
- 36. **Red spotted socks**: IgA deposition in skin in HSP causes ecchymoses and palpable purpura, especially in dependent areas (such as the legs, thighs, and buttocks)

- 37. **Henoch's kneeling pain**: IgA deposition in joints causes transient, oligoarticular (few joints), NON-deforming arthritis, especially in joints of the lower extremity (knees, ankles, feet)
- 38. **Kid hit in stomach**: HSP causes intermittent, colicky abdominal pain due to edema and hemorrhage of the intestinal submucosa caused by IgA deposition
- 39. **Red scorecard stain**: HSP often causes mild GI bleeding→ positive fecal occult blood test (clinically significant hemorrhage is rare)
- 40. **Telescoping ball retriever** : mucosal edema caused by IgA deposition in HSP can serve as a lead point for intussusception→ severe abdominal pain and "currant jelly" stool
- 41. **Glomerular red straw**: HSP causes deposition of IgA in the mesangium of glomeruli→ glomerulonephritis resembling IgA nephropathy (hematuria and occasional proteinuria)
- 42. **Steaming ears and red stained pants**: adults with HSP have more severe renal involvement, resulting in hypertension, more severe hematuria, nephrotic syndrome, and renal failure
- 43. **Cold older woman**: cryoglobulinemia is a type III hypersensitivity reaction (immune complex deposition) causing vasculitis (typically in older women)
- 44. **IgM to IgG ball retriever** : cryoglobulins are immunoglobulins (usually IgM) that bind each other via Fc regions of IgG and precipitate in cold temperatures
- 45. **Golf ball deposition**: immune complex deposition in cryoglobulinemia occurs at cold temperatures, causing complement activation, inflammation, and tissue damage
- 46. **Hippo water hazard**: there is a high association between cryoglobulinemia and hepatitis C infection (as well as multiple myeloma, myeloproliferative diseases, and lupus)
- 47. **Guy in shark shirt holding hole 4 pin**: Goodpasture syndrome (antiglomerular basement membrane disease) is caused by autoantibodies directed against type IV collagen in glomerular basement membranes (type II hypersensitivity reaction)
- 48. Linear antibody fence: immunohistochemistry of glomeruli in anti-GBM disease shows LINEAR IgG deposition (unique to anti-GBM disease)
- 49. "Complimentary Drinks": immunohistochemistry of glomeruli in anti-GBM disease shows extensive C3 deposition
- 50. **Spitting red drink**: autoantibodies in Goodpasture syndrome damage type IV collagen of alveolar basement membranes→ alveolar necrosis, shortness of breath, and hemoptysis





- 1. False mucosal teeth: a false diverticulum is an outpouching of mucosa and submucosa only (versus a true diverticulum which is outpouching of mucosa, submucosa, muscularis propria, and adventitia)
- 2. **Zany Zenker's hoodie**: Zenker diverticulum is a false diverticulum in the posterior upper esophagus
- 3. "klds go for enjoyment": Zenker's diverticulum is due to weakness of the cricopharyngeus muscle in the Killian triangle (an area of the hypopharynx)
- 4. **Eating burning pizza**: Zenker's diverticulum may present with odynophagia (painful swallowing)
- 5. **Stinky cheese**: Zenker's diverticulum may present with halitosis (bad breath)
- 6. **Regurgitating food**: Zenker's diverticulum may present with regurgitation
- 7. **Protruding "HIT-A-Mouse" game**: in a HIATAL HERNIA, the stomach herniates into the thorax through the esophageal hiatus
- 8. **Sliding mouse-spring junction**: in a SLIDING hernia, the gastroesophageal junction herniates through the esophageal hiatus (a type of HIATAL HERNIA)
- 9. **2 protruding mice**: in a PARAESOPHAGEAL hernia, the gastric cardia herniates through the esophageal hiatus (sneaking up beside a normally placed esophagus)(a type of HIATAL HERNIA)
- 10. **Refluxing acid**: HIATAL HERNIAs present with gastroesophageal reflux (GERD)

- 11. **Marshmallow pit**: Mallory-Weiss tears (mucosal tears in the proximal stomach and distal esophagus)
- 12. Linear scratches on distal slide: Mallory-Weiss tears consist of linear longitudinal lacerations in the mucosa of the distal esophagus and proximal stomach
- 13. **Drinking at marshmallow pit**: Mallory-Weiss tears are often due to alcohol abuse (increased intragastric pressure from vomiting)
- 14. "Wrenching" at marshmallow pit: Mallory-Weiss tears (and Boerhaave syndrome) are caused by increased intragastric pressure during retching and vomiting
- 15. **Coughing-up red drink**: Mallory-Weiss tears present with mild to moderate hematemesis
- 16. **Spilling red drink**: Mallory-Weiss tears can present with moderate hematemesis
- 17. Kick to the stomach: Mallory-Weiss tears can cause epigastric pain
- 18. **Ruptured bounce house**: in Boerhaave syndrome, there is transmural rupture of the esophagus
- 19. "Wrenching" at marshmallow pit: Mallory-Weiss tears (and Boerhaave syndrome) are caused by increased intragastric pressure during retching and vomiting
- 20. **PeriSCOPE**: Boerhaave syndrome can be caused by esophageal endoscopy
- 21. **Weakened bounce house**: conditions that weaken the esophageal wall (e.g. infection, inflammation, structural defects) are risk factors for Boerhaave syndrome
- 22. **Air-puff induced chest injury**: Boerhaave syndrome causes severe sharp chest pain
- 23. **Spilling red juice**: Boerhaave syndrome causes severe hematemesis
- 24. **CRUNCHY chip**: Boerhaave syndrome can cause subcutaneous emphysema (palpation of chest causes "crunchy" sound
- 25. **Wet pleural shirt**: Boerhaave syndrome can cause pleural effusions (due to gastric contents in pleural space)
- 26. **SEPTIC manhole cover and flame bandana**: Boerhaave syndrome can cause fever, sepsis, and septic shock





- 27. Gargling: gastroesophageal reflux disease (GERD) (an imbalance between LES tone and intragastric pressure causes reflux of gastric acid into the esophagus)
- 28. Loose drawstring: decreased LES tone can cause GERD
- 29. Smoker: smoking can cause GERD (due to decreased LES tone)
- 30. Alcoholic: alcohol use can cause GERD (due to decreased LES
- 31. Coffee: caffeine can cause GERD (due to decreased LES tone)
- 32. Pregnant woman: pregnancy can cause GERD (due to decreased LES tone and increased intragastric pressure)
- 33. Obese guy: obesity can cause GERD (due to increased intragastric
- 34. Punching chest: GERD can present with noncardiac chest pain such as heartburn or indigestion
- 35. Coughing in bed: GERD can present with nocturnal cough
- 36. Inhaler: GERD can present with nocturnal asthma
- 37. Choking throat: GERD can present with dysphagia (difficulty swallowing
- 38. Burning hole: GERD can progress to erosive esophagitis or esophageal ulcers
- 39. Clutching throat: erosive esophagitis due to GERD can present with odynophagia (painful swallowing)
- 40. Mucosal cake with pink and blue sprinkles: severe GERD causes esophagitis (on histology, appears as eosinophils and lymphocytes infiltrating the mucosa)
- 41. Tall lower cake layer with thick frosting: severe GERD causes elongation of the papillae of the lamina propria and hypertrophy of the basal cells of the mucosa

- 42. Barrett's bear: GERD can lead to Barrett's esophagus (intestinal metaplasia of the distal esophagus)
- 43. Squamous tiles: the esophagus is normally lined with stratified squamous epithelium
- 44. Columnar stage: intestinal metaplasia of Barrett's esophagus consists of columnar epithelium
- 45. **Scattered mucus cups**: intestinal metaplasia of Barrett's esophagus contains goblet cell hyperplasia
- 46. Metal intestines: intestinal metaplasia (Barrett's esophagus)
- 47. Ascending red velvety curtains: in Barrett's esophagus, patches of red velvety mucosa (columnar epithelium) extend upward from the gastroesophageal junction
- 48. Dirty dysplastic floor: dysplasia is a precancerous condition (compared to metaplasia, which is benign transformation of one cell type into another)
- 49. Cancer crab: Barrett's esophagus (METAplasia) can progress to DYSplasia, and eventually cancer (esophageal adenocarcinoma)
- 50. Parasol: GERD is treated with proton pump inhibitors (PPIs, suffix "prasol") or H2 blockers (both reduce acid production in the stomach)
- 51. Eosinophilic bilobed sling-shot: eosinophilic esophagitis presents similarly to severe GERD (dysphagia, chest/upper abdominal pain after
- 52. Slingshot kid: eosinophilic esophagitis often occurs in childhood (unlike GERD, which usually occurs in adults
- 53. Pink eosinophilic granules: eosinophilic esophagitis involves epithelial infiltration of eosinophils throughout the esophagus
- 54. Itchy nose and hives: eosinophilic esophagitis may present along with other atopic symptoms (eczema, allergic rhinitis, asthma, peripheral eosinophilia)
- 55. Breaking through parasol: Eosinophilic esophagitis may be resistant to PPIs

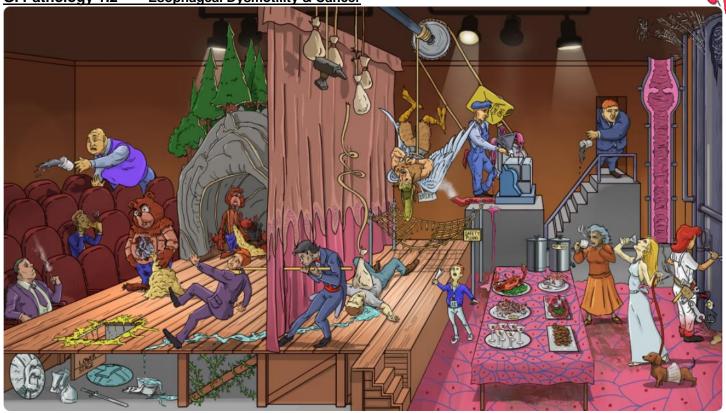
GI Pathology 1.2 Esophageal Dysmotility & Cancer



- 1. **Hooked at the throat**: ESOPHAGEAL CANCER and MOTILITY DISORDERS all present with DYSPHAGIA (difficulty swallowing)
- 2. Cancer crabs in bear den : esophageal ADENOCARCINOMA (the most common esophageal malignancy in the US) $\,$
- 3. **Glandular fluff**: esophageal ADENOCARCINOMA has gland-like features on histology (similar to other adenocarcinomas)
- 4. Barrett's bears : Barrett's esophagus can cause esophageal ADENOCARCINOMA (via progression to dysplasia \rightarrow cancer)
- 5. **Metal intestines**: Barrett's esophagus is metaplasia from normal stratified squamous epithelium to intestinal columnar epithelium
- Smoker: smoking increases the risk of esophageal ADENOCARCINOMA (especially if pre-existing Barrett's esophagus)
- 7. **Distal velvety curtains**: esophageal ADENOCARCINOMA primarily affects the distal third of the esophagus (including GE junction)(same area as GERD and Barrett's esophagus)
- 8. Stuck SOLID guy with LIQUID passing by: esophageal ADENOCARCINOMA presents with dysphagia (initially for SOLIDS, then also LIQUIDS)
- 9. Thin bear : esophageal ADENOCARCINOMA presents with weight loss
- 10. **Swallowing burning hot pizza**: esophageal ADENOCARCINOMA presents with odynophagia (painful swallowing)
- 11. Cancer crab on squamous tablecloth : esophageal SQUAMOUS cell carcinoma(the most common type of esophageal cancer worldwide)
- 12. **Cured foods**: foods containing N-nitroso compounds (pickled vegetables, preserved meats) are a risk factor for esophageal SQUAMOUS cell carcinoma (due to irritant effect)
- 13. **Hot tea**: hot beverages are a risk factor for esophageal SQUAMOUS cell carcinoma (due to irritant effect)
- 14. **Chewable nut**: betel nuts are a risk factor for esophageal SQUAMOUS cell carcinoma (due to irritant effect)
- 15. **Alcoholic**: alcohol is a risk factor for esophageal SQUAMOUS cell carcinoma (due to irritant effect)
- 16. **Smoker**: tobacco smoke is a risk factor for esophageal SQUAMOUS cell carcinoma (due to irritant effect)

- 17. **Mid-body tutu**: esophageal SQUAMOUS cell carcinoma usually occurs in the middle third of the esophagus
- 18. **Stuck SOLID guy with LIQUID passing by**: esophageal SQUAMOUS CELL CARCINOMA presents with dysphagia (initially for SOLIDS, then also LIQUIDS)
- 19. **Clutching throat**: esophageal SQUAMOUS cell carcinoma causes odynophagia (painful swallowing)
- 20. **Thin actress**: esophageal SQUAMOUS cell carcinoma causes weight loss
- 21. Raised flat area: esophageal SQUAMOUS cell carcinoma appears as raised plaques with ulceration
- 22. **Pearl necklace**: on histology esophageal SQUAMOUS cell carcinoma displays keratin pearls and intercellular bridging (similar to other forms of squamous cell carcinoma)
- 23. **Plumber**: Plummer-Vinson syndrome (triad of dysphagia, iron deficiency anemia, and esophageal webs)
- 24. Falling iron tools: PLUMMER-VINSON syndrome is caused by chronic iron deficiency
- 25. **Cobwebs**: PLUMMER-VINSON syndrome causes esophageal webs
- 26. **Mucosal protrusions**: esophageal webs in PLUMMER-VINSON syndrome are thin protrusions of esophageal mucosa in the upper third of the esophagus → dysphagia for SOLIDS only
- 27. Stuck SOLID guy : PLUMMER-VINSON syndrome presents with dysphagia for SOLIDS
- 28. **Pale plumber's overalls**: PLUMMER-VINSON syndrome presents with iron deficiency anemia
- 29. Irritated squamous tile: PLUMMER-VINSON syndrome increases the risk of esophageal SQUAMOUS cell carcinoma (due to irritant effect)

GI Pathology 1.2 Esophageal Dysmotility & Cancer



- 30. **Squeezing lower esophageal harness**: ACHALASIA is a esophageal DYSMOTILITY disorder caused by impaired relaxation of the LES → functional esophageal obstruction)
- 31. Loss of safety plexus : ACHALASIA is caused by impaired relaxation of the LES due to loss of the myenteric (Auerbach) plexus
- 32. Rusty nitric oxide exhaust & falling VIP pass : in ACHALASIA, loss of the myenteric (Auerbach) plexus \rightarrow decreased production of NO and VIP \rightarrow impaired relaxation of the LES smooth muscle
- 33. "Idiot": ACHALASIA is usually idiopathic
- 34. **Che's Gas**: Acquired ACHALASIA is caused by Chagas disease (Trypanosoma cruzi destroys myenteric plexus)
- 35. **Bird beak**: in ACHALASIA, increased LES tone leads to constriction of the LES and dilation of the esophagus more proximally (appears as bird-beak on barium swallow)
- 36. $\mbox{\bf Regurgitating food}: \mbox{ACHALASIA presents with regurgitation of undigested food}$
- 37. Clutching chest: ACHALASIA presents with chest pain
- 38. **Difficulty funneling liquid**: ACHALASIA presents with dysphagia for LIQUIDS (in addition to SOLIDS
- 39. **Uncoordinated spasming rope**: DIFFUSE ESOPHAGEAL SPASM is a DYSMOTILITY disorder (in which impaired inhibitory neurotransmission within the myenteric plexus → diffuse uncoordinated contractions)
- 40. "Corkscrew" rope : DIFFUSE ESOPHAGEAL SPASM appears as a "corkscrew" on barium swallow
- 41. **Regurgitating**: DIFFUSE ESOPHAGEAL SPASM causes regurgitation
- 42. **Leaking water**: DIFFUSE ESOPHAGEAL SPASM causes dysphagia for LIQUIDS (in addition to SOLIDS
- 43. Sand bag crushing chest: DIFFUSE ESOPHAGEAL SPASM causes retrosternal chest pain
- 44. **Anginal anvil**: in DIFFUSE ESOPHAGEAL SPASM, chest pain is similar to angina (and also relieved by nitrates)

- 45. **Dragon talon crest**: CREST syndrome (Calcinosis, Raynaud syndrome, Esophageal dysmotility, Sclerodactyly, Telangiectasias) is a variant of systemic sclerosis (autoimmune disease targeting vascular endothelium, causes tissue fibrosis)
- 46. **Fibrous vines in lower stage**: in CREST syndrome, esophageal dysmotility is due to fibrous replacement of the muscularis in the lower esophagus
- 47. **Dripping water**: CREST syndrome presents with dysphagia for LIQUIDS (in addition to SOLIDS)
- 48. **Refluxed acid**: in CREST syndrome, decreased esophageal tone can lead to GERD (increasing risk of Barrett's esophagus)

GI Pathology 1.3 Acute and Chronic Gastritis & Peptic Ulcer Disease



- 1. Damaged peptic concrete: acute gastropathy (e.g. damaged gastric mucosa due to deficient mucus lining or direct toxic effect)
- 2. Fire extinguisher : NSAIDs can cause acute gastropathy (direct toxic effect; AND inhibition of COX-1 \rightarrow decreased prostaglandin synthesis \rightarrow decreased mucus production)
- 3. **Smoking and drinking**: both alcohol and tobacco have direct toxic effects on the gastric epithelium, which can lead to acute gastropathy
- 4. Flaming peptic concrete: acute gastropathy can lead to acute gastritis (vascular congestion, edema, and neutrophilic infiltration on histology)
- 5. **Leaking shallow hole**: severe damage may lead to an EROSION which extends through the gastric epithelium into the lamina propria (may cause acute hemorrhagic erosive gastropathy)
- 6. Leaking deep hole through rebar layer: gastric ulcers are erosions that have extended through the muscularis mucosa into the submucosa (risk for bleeding)
- 7. Burning curly drill bit: Curling ulcers can develop with severe burns
- 8. **Dumping bucket on burn**: Curling ulcers develop due to hypovolemia and decreased gastric mucosal perfusion
- 9. **Crushing head**: Cushing ulcers can develop with high intracranial pressure
- 10. **Vegas sign**: increased intracranial pressure causes massive vagal output \to increased gastric acid production \to Cushing ulcers
- 11. **Stomach on fire** : acute gastritis can cause vague epigastric pain (dyspepsia)
- 12. Nauseated : acute gastritis can cause nausea, vomiting, anorexia
- 13. **Chronic grandfather clock** : chronic gastritis (e.g. H. pylori infection, autoimmune gastritis)
- 14. **Helicopter**: H. pylori infection is the most common cause of chronic gastritis
- 15. **Helicopter sinking through mucus**: H. pylori invades the stomach's mucus layer to reside on the gastric epithelium
- 16. Pink commas: H. pylori is a comma-shaped gram-negative bacilli
- 17. Helicopter adjacent to distal stomach: H. pylori usually infects the antrum first
- 18. Fire under helicopter: initial infection with H. pylori can cause acute gastritis

- 19. **Spotlight pointing across the stomach**: H. pylori infection spreads from the antrum to the body of the stomach
- 20. Chronic shack roof on fire : chronic H. pylori infection causes inflammation of gastric mucosa
- 21. **First responders**: chronic inflammation due to H. pylori infection displays a prominent neutrophil infiltrate
- 22. **Thinning fabric**: chronic inflammation from H. pylori leads to chronic atrophic gastritis (mucosal atrophy and loss of acid- and hormone-secreting glands → decreased gastrin and acid secretion)
- 23. White mucus knots in columnar fence: chronic inflammation from H. pylori leads to intestinal metaplasia (normal gastric columnar epithelium develops goblet cell hyperplasia)
- 24. **Intestinal metal pipes** : chronic inflammation from H. pylori leads to intestinal metaplasia
- 25. Cancer crab; intestinal metaplasia (due to chronic inflammation from H. pylori) can progress to dysplasia and cancer:
- 26. **Pile of lymphoid tissues**: chronic H. pylori infection can lead to development of Mucosa-Associated Lymphoid Tissue (MALT) (chronic antigenic presence of H. pylori causes accumulation of lymphocytes, which organize into lymphoid tissue with germinal centers)
- 27. **Blue tissues**: on histology, MALT appear blue due to the presence of lymphocytes
- 28. **Crab on helicopter**: MALT (caused by H. pylori infection) can transform into gastric lymphoma
- 29. **Biopsy grabber**: H. pylori infection can be diagnosed with gastric biopsy (antrum and body)
- 30. **Ammonia spray bottle**: urease testing of gastric biopsy specimen increases sensitivity of testing for H. pylori
- 31. **Gas from ammonia bottle**: H. pylori can be diagnosed with a urea breath test (patient drinks radiolabeled urea → urease in H. pylori metabolizes urea into radiolabeled CO2 → patient breathes CO2 into a detector)

GI Pathology 1.3 Acute and Chronic Gastritis & Peptic Ulcer Disease



- 32. **Antibody forklift**: in autoimmune metaplastic atrophic gastritis, autoantibodies cause destruction of parietal cells and intrinsic factor
- 33. Targeting parietal protesters: parietal cells (located in body of stomach) are targeted in autoimmune metaplastic atrophic gastritis
- 34. **Dropping lemons**: in autoimmune metaplastic atrophic gastritis, destruction of parietal cells causes decreased secretion of gastric acid
- 35. **Oversized "GAS" can**: in autoimmune metaplastic atrophic gastritis, gastrin levels are increased (destruction of parietal cells \rightarrow decreased HCl secretion \rightarrow hyperplasia of gastrin-producing G cells in antrum \rightarrow hypergastrinemia)
- 36. "NO INTRINSIC right": in autoimmune metaplastic atrophic gastritis, intrinsic factor is decreased (normally binds to vitamin B12 to facilitate its absorption)
- 37. **Mushroom cloud "blast"**: in autoimmune metaplastic atrophic gastritis, decreased B12 absorption can cause pernicious anemia (a megaloblastic anemia [large, fragile erythrocytes])
- 38. **Jackhammer eroding through rebar layer**: peptic ulcer disease (PUD) (ulcers penetrate through the muscularis mucosa into the submucosa)
- 39. **P.U.D. helicopter**: PUD usually develops over a background of chronic gastritis (e.g. H. pylori infection)
- 40. Lesser curvature hole: gastric ulcer (most commonly occur in the lesser curvature of the stomach)
- 41. **Distal duodenal hole** : duodenal ulcer (more common than gastric ulcer)
- 42. **Knocked-over STOP sign**: H. pylori-mediated chronic gastritis leads to the destruction of antral delta cells → decreased somatostatin
- 43. **GAS tanks**: in chronic gastritis (e.g. H. pylori infection), loss of somatostatin leads to uninhibited gastrin production
- 44. Activated protesters throwing lemons at gas guy: increased gastrin stimulates acid secretion by parietal cells
- 45. **Lemon juice in duodenal hole**: in chronic gastritis (e.g. H. pylori infection), increased acid delivery to the duodenum leads to duodenal erosion and ulcers
- 46. **Fire extinguisher**: NSAID use is a risk factor for peptic ulcer disease in the STOMACH (SYNERGISTIC with H. Pylori)

- 47. **Smoker**: smoking is a risk factor for peptic ulcer disease (both gastric and duodenal)
- 48. Eating lunch with active jackhammer: in GASTRIC peptic ulcer disease, epigastric pain is WORSE with meals (due to increased gastrinmediated acid secretion)
- 49. **Eating lunch with inactive jackhammer**: in DUODENAL peptic ulcer disease, epigastric pain is IMPROVED with meals (due to decreased passage of acid into the duodenum after food intake, and release of bicarbonate from the pancreas)
- 50. **Hole leaking blood**: peptic ulcer disease can cause upper Gl bleeding (e.g. melena or hematemesis if severe)
- 51. **Black tar**: peptic ulcer disease can cause melena ("black tarry stools" due to oxidation of iron from hemoglobin breakdown), a complication of UPPER GI bleeds (proximal to suspensory ligament of the duodenum)
- 52. **Big gastric blood pipe**: gastric ulcers may bleed heavily (due to erosion into left gastric artery)
- 53. "Path Obstructed": gastric outlet obstruction is a complication of peptic ulcer disease (due to inflammation causing swelling of pylorus)(presents with nonbilious emesis)
- 54. **Big duodenal air pipe**: duodenal ulcers can lead to perforation (most common in anterior duodenum)
- 55. **Small gastric air pipe**: gastric ulcers can lead to perforation (less common than in duodenal ulcers)
- 56. **Suspender strap popping** : ulcer perforation causes shoulder pain (acute peritonitis \rightarrow irritation of diaphragm and phrenic nerve \rightarrow pain in C3-C5 dermatomes)
- 57. **Black belt**: ulcer perforation causes radiolucency (i.e. darkness) between right hemidiaphragm and liver on upright abdominal X-ray (due to free air in abdominal cavity)
- 58. **Grabbing crab**: peptic ulcers and surrounding areas are biopsied to rule-out malignancy

GI Pathology 1.4 Gastric Dysmotility & Cancer





- 1. **Broken gastric rickshaw**: gastroparesis (delayed gastric emptying WITHOUT mechanical obstruction)
- 2. Nauseated: gastroparesis presents with nausea and vomiting
- 3. Holding abdomen: gastroparesis presents with abdominal pain
- 4. **Broken "VEGAS" sign** : gastroparesis is caused by dysfunction of the vagus nerve (parasympathetic nervous system) → impaired peristalsis of food through stomach
- 5. **Severed wire**: iatrogenic effects (e.g. surgical injury to the vagus nerve) can cause gastroparesis
- 6. "IDIOT": gastroparesis is most commonly idiopathic
- 7. Elevated candy: diabetes can cause gastroparesis
- 8. **Mortar and pestle**: medications (e.g. calcium channel blockers, tricyclic antidepressants) can cause gastroparesis
- 9. "raMEN TRy It": Menetrier's disease (hypertrophy of stomach rugal folds due to hyperplasia of mucus-secreting cells)
- 10. **Thick ramen noodles**: in Menetrier's disease, hypertrophy of rugal folds inhibits peristalsis of food through the stomach
- 11. Excessive mucus : in Menetrier's disease, hyperplasia of mucus-secreting cells results in decreased number of acid-secreting gastric glands \rightarrow decreased protein digestion
- 12. Nauseated: Menetrier's disease presents with nausea and vomiting
- 13. **Holding abdomen**: Menetrier's disease presents with abdominal pain and early satiety
- 14. Tarry black broth : Menetrier's disease can cause melena and hematemesis (symptoms of upper GI bleeding)
- 15. Losing meat chunks: Menetrier's disease is a "protein-losing enteropathy" (due to increased permeability of stomach mucosal cells)
- 16. Thin girl: Menetrier's disease presents with weight loss, muscle wasting, hypoalbuminemia, peripheral edema (due to protein loss)
- 17. Cancer crab : Menetrier's disease is a risk factor for gastric adenocarcinoma
- 18. **Giant cancer crab** : gastric adenocarcinoma (the most common malignancy of stomach, more common in East Asian countries)
- 19. **Hanging intestines**: intestinal-type gastric adenocarcinoma (the most common gastric adenocarcinoma)
- 20. **Grandfather clock** : chronic atrophic metaplastic gastritis is the biggest risk factor for intestinal-type gastric adenocarcinoma
- 21. **Helicopter fan**: H. pylori-mediated chronic gastritis is a risk factor for intestinal-type gastric adenocarcinoma
- 22. **Antibody on gastric pot**: autoimmune gastritis is a risk factor for intestinal-type gastric adenocarcinoma
- 23. **Hanging meats**: consumption of salt-preserved foods and nitrosamine-preserved foods are risk factors for gastric adenocarcinoma
- 24. **Smoking & drinking**: smoking and drinking are risk factors for gastric adenocarcinoma

- 25. "Epstein's Bar" : Epstein-Barr virus (EBV) infection is a risk factor for gastric adenocarcinoma
- 26. **Cauliflower**: intestinal-type gastric adenocarcinoma forms a bulky exophytic mass (occurs in lesser curvature of the antrum and pylorus)
- 27. **Oozing red sauce**: intestinal-type gastric adenocarcinoma may cause bleeding and ulcerations
- 28. **Mucinous rice**: on histology, intestinal-type gastric adenocarcinoma displays glandular structures filled with mucin
- 29. "Claw-adhering" rubber band : E-cadherin (an intercellular adhesion molecule)
- 30. **Losing "claw-adhering" band**: in diffuse-type gastric adenocarcinoma, loss of normal E-cadherin proteins results in decreased intercellular connections → disorganized cell growth
- 31. **Crabs infiltrating along wall**: in diffuse-type gastric adenocarcinoma, disorganized cells growth leads to diffuse invasion throughout stomach ("infiltrative growth pattern")
- 32. **Leather stomach purse**: diffuse-type gastric adenocarcinoma causes a fibrosing desmoplastic reaction → linitis plastica ("leather bottle" stomach)
- 33. Ring jewelry: on histology, diffuse-type gastric adenocarcinoma cells display "signet rings" (mucin-containing vacuoles displace nucleus to cell membrane)
- 34. Rings on liver purse, lapels, and pockets: gastric adenocarcinomas (intestinal and diffuse types) most commonly metastasize to liver, lungs, and ovaries
- 35. Chess board with white T knights & B antibody archers : primary gastric lymphomas (T or B-cell)
- 36. ${\bf Grandfather\ clock}$: chronic atrophic metaplastic gastritis is a risk factor for gastric lymphoma
- $\textbf{37. Helicopter-like fan}: \textbf{H. pylori-mediated chronic gastritis is a risk factor for gastric lymphoma \\$
- 38. Pile of blue lymphoid tissues: H. pylori-mediated chronic gastritis induces MALT which can progress to gastric lymphoma
- 39. Epstein's Sake Bar : EBV infection can cause gastric lymphoma
- 40. Marginalized B archers & large B archers: gastric lymphomas are most commonly marginal zone B cell or diffuse large B cell lymphoma
- 41. "GIST" arcade hall: GI stromal tumor (GIST mesenchymal tumor, derived from interstitial cells of Cajal)
- 42. **Rhythm game**: GIST is derived from the interstitial cells of Cajal (GI "pacemaker" cells, responsible for peristalsis)]
- 43. Tool kit: GIST is caused by gain-of-function mutations in the c-KIT oncogene
- 44. Tire swing game: C-kit is a tyrosine kinase
- 45. **Under mucosal door** : GISTs form well-circumscribed, fleshy, submucosal masses
- 46. **Mushroom family**: familial adenomatous polyposis (FAP causes colon polyps, but no gastric polyps) is a risk factor for gastric adenocarcinoma

GI Pathology 2.1 Small Bowel Obstruction, Ileus & Hernias



- 1. OBSTRUCTED by rock debris: small bowel obstruction
- 2. Latching on to shirt: adhesions are the most common cause of small bowel obstruction (due to mechanical constriction of bowel)
- 3. **Scalpel graffiti**: prior abdominal surgery is the most common cause of abdominal adhesions
- 4. Cancer crab skateboard : small bowel tumors can cause obstruction
- 5. Green face : small bowel obstruction presents with nausea/vomiting
- 6. Collie: small bowel obstruction presents with colicky abdominal pain
- 7. **Plunger**: small bowel obstruction presents with obstipation (inability to pass flatus or stool)
- 8. Large belly: small bowel obstruction presents with distended abdomen (due to backup of gas in the intestines)
- 9. **Drum**: small bowel obstruction presents with tympanitic abdomen (due to backup of gas in the intestines)
- 10. "Tink, tink!": small bowel obstruction presents with high-pitched "tinkling" bowel sounds on abdominal auscultation (due to abnormal peristalsis)
- 11. **Black cloud graffiti**: on X-ray, small bowel obstruction displays bowel distension (dilated loops of bowel) and air-fluid levels (horizontal contrast line)
- 12. **Wide to narrow half-pipe transition**: on X-ray, small bowel obstruction displays a "transition point" (location of obstruction, point where dilated proximal bowel meets normal distal bowel)
- 13. **Broken skateboard**: ileus is caused by hypomotility (similar symptoms as small bowel obstruction)
- 14. **Dilated colon pants**: in ileus, bowel is uniformly dilated (including colon and rectum)
- 15. **Broken boombox**: ileus presents with diminished or absent bowel sounds (due to decreased peristalsis)
- 16. Scalpel graffiti : surgery can cause ileus
- 17. Drug bottles: drugs can cause ileus
- 18. SEPTIC cover: sepsis can cause ileus
- 19. **Elevated candy jar**: diabetes can cause ileus (due to dysfunctional parasympathetic nervous system)
- 20. Arterial rail: superior mesenteric artery (SMA)

- 21. Red ramp: aorta
- 22. **Crushed kid**: in SMA syndrome, the third portion of the duodenum is compressed between the aorta and SMA (can cause small bowel obstruction)
- 23. **Losing padding**: SMA is caused by severe rapid weight loss (shrinking mesenteric fat pad → decreased cushioning around duodenum → duodenal compression)
- 24. **Painful burrito**: SMA syndrome presents with recurrent postprandial abdominal pain
- 25. **Telescoping camera lens**: intussusception (occurs when segment of proximal bowel telescopes beneath more distal bowel)
- 26. **Skull shirt**: intussusception can cause bowel ischemia and necrosis (due to impaired venous return)
- 27. **Pile of lymphoid tissues**: hyperplastic lymphoid tissue (usually post-viral infection) can serve as a lead point to initiate intussusception (usually in toddlers)
- 28. **Meckel's hecklers**: Meckel's diverticulum can serve as a lead point to initiate intussusception
- 29. **Mushroom hats**: polyps and tumors can serve as a lead point to initiate intussusception
- 30. **Small ramp meets large landing zone** : the ileocecal junction is the most common site for intussusception
- 31. **Gooey red jelly**: intussusception presents with "currant jelly" stools (blood and mucus)
- 32. **Handlebar at RLQ**: intussusception presents with right lower quadrant mass (location of ileocecal junction)
- 33. **Target cushion**: on abdominal ultrasound, intussusception has a "target" appearance (due telescoped bowel)
- 34. "BERRY-YUM": barium enema can diagnose and treat intussusception (moves telescoped bowel)

GI Pathology 2.1 Small Bowel Obstruction, Ileus & Hernias



- 35. Weakness in chain link fence : hernias occur as a results of weakening of fibromuscular tissues \rightarrow intra-abdominal contents protrude through defect
- 36. Running: strenuous exercise is a risk factor for hernias
- 37. **Tight abdominal belt**: chronic elevated intra-abdominal pressure (i.e. heavy lifting, chronic cough, constipation, pregnancy) is a risk factor for hernia
- 38. **Older male**: hernias are more common older age (decreased tissue strength) and men
- 39. **Handcuffs**: incarcerated hernia (abdominal contents [omentum, small bowel, colon] unable to escape hernia)
- 40. **Constricted blue and red strings**: incarcerated hernias can cause constriction of veins within bowel \rightarrow decreased venous and lymphatic drainage \rightarrow swelling \rightarrow compromised arterial blood flow
- 41. Skull tattoos: bowel strangulation can lead to necrosis
- 42. **Torn pants**: bowel strangulation can lead to perforation
- 43. SEPTIC cover: bowel strangulation can lead to sepsis
- 44. **HESSELBACH'S construction**: Hesselbach's Triangle (bound by the inguinal ligament, rectus abdominis, and inferior epigastric vessels)
- 45. Tube: inguinal canal
- 46. **Tarp under skater tunnel**: inguinal ligament (base of inguinal canal) forms inferior border of Hesselbach's Triangle)
- 47. **Striated wooden wall**: rectus abdominus muscle (medial border of Hesselbach's Triangle)
- 48. **Arterial and venous tubes**: inferior epigastric vessels (lateral border of Hesselbach's Triangle)
- 49. **Directly pressing on fence**: DIRECT inguinal hernias pass DIRECTLY through Hesselbach's Triangle (MEDIAL to inferior epigastric vessels)

- 50. **Sneaking through tube**: INDIRECT inguinal hernias pass through inguinal canal (LATERAL to inferior epigastric vessels)
- 51. **Sneaking from under tarp**: FEMORAL hernias pass through femoral canal (located inferior-posterior to inguinal ligament)
- 52. **Shirt and hoodie**: INDIRECT inguinal hernias are covered by all three layers of spermatic fascia (external oblique aponeurosis, cremasteric fascia, transversalis fascia)
- 53. **Shirt only**: DIRECT inguinal hernias are covered only by external spermatic fascia
- 54. **Weakened fence**: DIRECT inguinal hernias are a result of weakness in the floor of the inguinal canal
- 55. "VIRGINIA Co PATENT": INDIRECT inguinal hernias are a result of a persistent PATENT processus vaginalis
- 56. **Woman with skull tattoo**: femoral hernias occur more frequently in women (high rate of incarceration and strangulation due to small opening of femoral ring)

GI Pathology 2.2 Carcinoid Tumor & Small Bowel Neoplasms



- 1. **Ooze canister** : carcinoid tumor (most common primary small bowel malignancy)
- 2. **Neural transformer**: carcinoid tumors are derived from neuroendocrine cells
- 3. **Ooze in distal intestinal pipes**: the ileum is the most common site for carrinoid tumors
- 4. Slime in gastric tunnel: carcinoid tumors can occur in the stomach
- 5. Slime in haustra tunnel: carcinoid tumors can occur in the colon
- 6. **Ooze in appendiceal pipe**: carcinoid tumors are the most common primary malignancy of the appendix
- 7. Chest ooze: carcinoid tumors can occur in the lungs ("bronchial carcinoid tumors")
- 8. **Child in the ooze**: bronchial carcinoid tumors are the most common primary lung cancer in children
- 9. **Coughing blood**: bronchial carcinoids present with coughing and hemoptysis
- 10. 10Yellow ooze : carcinoid tumors appear yellow on gross pathology
- 11. **Purple spots on pink**: on histology, carcinoid tumors contain islands of small round purple cells within pink connective tissue
- 12. **Granular granite**: on electron microscopy, carcinoid tumor cells display cytoplasmic granules (contain secretory products and hormones)
- 13. "Path Obstructed": small bowel carcinoid tumors may cause intestinal obstruction
- 14. **Inflamed appendiceal pipe**: carcinoid tumors of the appendix may cause appendicitis (due to obstruction of the appendiceal lumen)
- 15. Climbing into liver base : GI carcinoid tumors may metastasize to the liver
- 16. **Liver armor**: because of first pass metabolism in the liver, substances secreted by carcinoid tumors in the GI tract do not cause systemic symptoms
- 17. Consuming histamine bee : carcinoid tumors secrete histamine, which is metabolized by the liver
- 18. Consuming smiley serotonin pizza: carcinoid tumors secrete serotonin, which is metabolized by the liver
- 19. **Bunch of pizza and bees**: vasoactive substances (e.g. serotonin, histamine) cause symptoms ("carcinoid syndrome") if tumor metastasizes to liver or beyond GI tract (i.e. bypass first pass metabolism by the liver)
- 20. 20Red face: carcinoid syndrome causes episodic flushing (face, neck, chest)(due to excessive histamine secretion)
- 21. **Red face mask**: carcinoid syndrome causes vascular telangiectasias on nose and cheeks (due to vasodilation from vasoactive factors)
- 22. **Brown waterfall**: carcinoid syndrome can cause secretory diarrhea (excessive serotonin → increased intestinal motility)

- 23. **Wheezy party blower**: carcinoid syndrome may cause episodic bronchospasm and wheezing
- 24. **Slimy right-sided valves**: carcinoid syndrome can cause valvular disease (plaque-like vegetations) in the right heart (i.e. tricuspid and pulmonic valves)
- 25. **Smiley graffiti**: carcinoid valvular disease is due to due to excessive serotonin (causes increased fibroblast growth)
- 26. **Leaking wheel**: carcinoid valvular disease can cause regurgitation (may later progress to stenosis)
- 27. **Consuming serotonin smiley pizza**: the lungs metabolize serotonin (produced by carcinoid tumors)
- 28. Catching histamine bee : the lungs metabolize histamine (produced by carcinoid tumors)
- 29. **Lung chest armor**: the lungs metabolize vasoactive substances secreted by carcinoid tumors (including serotonin, therefore no serotonin reaches left heart \rightarrow no left heart valvular disease)
- 30. 30 "HI-AA!": carcinoid syndrome is diagnosed with urine 5-HIAA (5-hydroxyindoleacetic acid)(a breakdown product of serotonin)
- 31. **STOP sign** : somatostatin analogues are used to treat carcinoid syndrome (bind to somatostatin receptors → inhibit release of serotonin)
- 32. 8 sided sign: OCTreotide is a somatostatin analogue
- 33. Super pincher crab: small bowel adenocarcinoma
- 34. **Glandy bubbles**: on histology, small bowel adenocarcinoma displays glandular structures
- 35. **Proximal duodenal pipe**: the duodenum is the most common site for small bowel adenocarcinomas
- 36. **Mutated shroom family**: familial adenomatous polyposis (FAP, causes polyps in duodenum and colon) is a risk factor for small bowel adenocarcinoma
- 37. **Inch worm**: Lynch syndrome is a risk factor for small bowel adenocarcinoma
- 38. Peutz pop : Peutz-Jeghers syndrome
- 39. **Hammers**: Peutz-Jeghers syndrome causes multiple non-neoplastic HAMARTOMATOUS polyps throughout GI system (mostly small bowel)
- 40. Domino: Peutz-Jeghers syndrome is autosomal dominant
- 41. **Creeping crab**: Peutz-Jeghers syndrome is a risk factor for small bowel adenocarcinoma (as well as colorectal and non-GI cancers)
- 42. Pigmented Peutz pop syrup: Peutz-Jeghers syndrome causes mucocutaneous hyperpigmentation (mouth, lips, palms, genitalia)
- 43. **Skipping cobble stones**: Crohn's disease is a risk factor for small bowel adenocarcinoma
- 44. 44**Distal pipe**: Crohn's disease (and resulting small bowel adenocarcinoma) commonly affects the ileum

GI Pathology 2.3 Intestinal Ischemia & Angiodysplasia



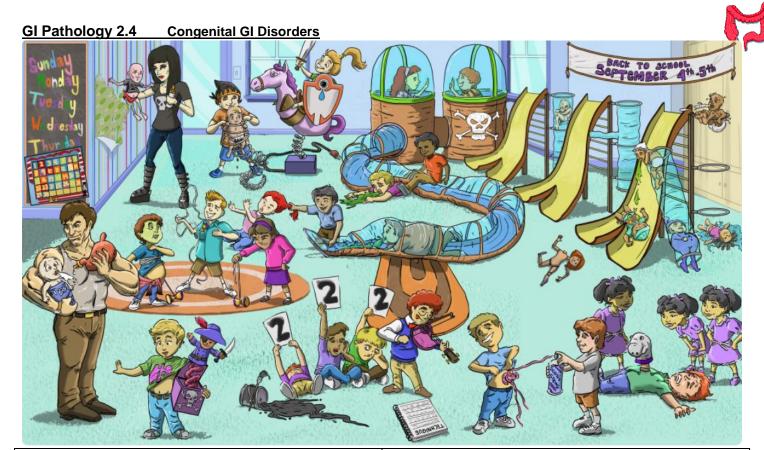
- 1. Day of the Dead: intestinal ischemia and necrosis (occurs as a result of inadequate perfusion of the bowel)
- 2. Tall tombstone projections with vascular ivy: villi of small and large bowel (contain small capillaries and venules)
- 3. "Crypt": crypts of small and large bowel (contain secretory and stem cells)
- 4. Small vascular ivy near surface : arteries become smaller (and more susceptible to ischemia) as they move toward the luminal surface of the villi
- 5. Cracking dead vascular ivy: bowel ischemia and necrosis start at the luminal surface of villi \to leads to sloughing of villi
- 6. **Proliferating crypt flowers**: in the bowel, crypts are initially spared from ischemia and become hyperplastic (although later affected as ischemia progresses)
- 7. First responders: ischemia leads to a neutrophilic infiltrate
- 8. **Patches of red marigolds**: bowel ischemia leads to patchy areas of mucosal hemorrhage
- 9. **High pressure water** : ischemia and inflammation lead to bowel wall EDEMA \rightarrow increased pressure obstructs venous drainage \rightarrow further decrease in perfusion
- 10. **Top arterial bowsprit**: superior mesenteric artery (SMA)(supplies small intestines and proximal large bowel)
- 11. Low arterial bowsprit: inferior mesenteric artery (IMA)(supplies distal large bowel)
- 12. **Black intestinal design**: ACUTE INTESTINAL OCCLUSION (most likely to occur in the SMA, leads to ACUTE MESENTERIC ISCHEMIA)
- 13. Poop emboli from thrombotic nest: occlusion of the SMA by an EMBOLUS causes ACUTE MESENTERIC ISCHEMIA
- 14. Irregularly irregular TV signal: atrial fibrillation can cause ACUTE MESENTERIC ISCHEMIA (via embolus formation)
- 15. **Thrombotic straw dress**: thrombus formation in the SMA (due to rupture of atherosclerotic plaques) causes ACUTE MESENTERIC ISCHEMIA
- 16. **Abdominal pain perplexing doctor**: acute mesenteric ischemia causes severe abdominal pain (out of proportion to physical exam)
- 17. Green face: ACUTE MESENTERIC ISCHEMIA causes nausea/vomiting
- 18. **Broken speaker**: ACUTE MESENTERIC ISCHEMIA causes loss of bowel sounds (due to ileus)
- 19. **Dressed in all black with ripped sleeve**: ACUTE MESENTERIC ISCHEMIA can lead to bowel infarction and perforation
- 20. "SEPTIC: ACUTE MESENTERIC ISCHEMIA can lead to sepsis
- 21. "Open" door: NON-occlusive acute intestinal ischemia
- 22. **Hypotensive fainting**: NON-occlusive acute intestinal ischemia is caused by hypoperfusion of intestinal vessels
- 23. "Water Shed": the colon has WATERSHED areas (areas most susceptible to hypoperfusion)
- 24. Arterial trees barely reaching each other: arteries in the colon connect with each other via small shared vessels (anastomoses)

- 25. Losing water: hypovolemia can cause ACUTE COLONIC ISCHEMIA (because of poor perfusion)
- 26. SEPTIC: septic shock can cause ACUTE COLONIC ISCHEMIA (because of poor perfusion)
- 27. **Heart shirt** : cardiogenic shock can cause ACUTE COLONIC ISCHEMIA (because of poor perfusion)
- 28. Elderly: ACUTE COLONIC ISCHEMIA most commonly occurs in the elderly)
- 29. Corner tree attachment: watershed area between SMA and IMA (located at the splenic flexure, where the transverse colon meets the descending colon)
- 30. **Distal tree attachment**: watershed area between IMA and rectal arteries (located at the distal end of sigmoid colon)
- 31. **Clutching left abdomen**: ACUTE COLONIC ISCHEMIA causes crampy LEFT abdominal pain and tenderness (watershed areas are on the left side)
- 32. Red blood puddle: ACUTE COLONIC ISCHEMIA can cause bloody stools (due to necrosis and hemorrhage)
- 33. **Broken speaker**: ACUTE COLONIC ISCHEMIA can cause loss of bowel sounds (due to ileus)
- 34. SEPTIC: ACUTE COLONIC ISCHEMIA can cause sepsis
- 35. Anvil: CHRONIC MESENTERIC ISCHEMIA causes "intestinal angina"
- 36. Grandfather clock: chronic
- 37. Greasy pipe: CHRONIC MESENTERIC ISCHEMIA (occurs secondary to atherosclerosis of mesenteric vessels
- 38. Obese grandma : CHRONIC MESENTERIC ISCHEMIA is most common in obese older females
- 39. **Greasy spatula**: other atherosclerosclerotic disease (coronary heart disease, cerebrovascular disease, peripheral vascular disease) is a risk factor for CHRONIC MESENTERIC ISCHEMIA
- 40. **Clutching belly**: CHRONIC MESENTERIC ISCHEMIA presents with postprandial abdominal pain (due to increased oxygen demand during digestion)
- 41. Aversion to burrito: CHRONIC MESENTERIC ISCHEMIA presents with aversion to eating (due to postprandial abdominal pain)
- 42. Thin skeleton: CHRONIC MESENTERIC ISCHEMIA presents with weight loss (due to aversion to eating)
- 43. **Tangled red strings**: ANGIODYSPLASIA of the GI tract (caused by malformed blood vessels [thin-walled, dilated, tortuous, composed only of endothelium])
- 44. **Colonic dress**: ANGIODYSPLASIA most commonly occurs in the colon (particularly the cecum)
- 45. **Pool of blood**: ANGIODYSPLASIA presents with recurrent, painless hematochezia
- 46. Stenotic arched hat : bleeding of ANGIODYSPLASIA is associated with aortic arch stenosis



- 1. Pinched stomach bottle: pyloric stenosis
- 2. **#1 baby boy** : pyloric stenosis is more common in males (especially first-born males)
- 3. **Vomiting white milk**: pyloric stenosis presents with non-bilious (because proximal to duodenum) projectile vomiting immediately after feeding
- 4. One month calendar : pyloric stenosis presents ~ 1 month after birth (4th-5th week of life)
- 5. **Muscular babysitter**: pyloric stenosis is caused by hypertrophy of the pyloric sphincter (takes ~ 1 month to develop)
- 6. Round bottle nipple: pyloric stenosis may present with a palpable "olive-shaped" mass at the pylorus (periumbilical)
- 7. **Schisis string next to umbilicus**: gastroschisis (paraumbilical herniation of the abdominal contents through the ventral abdominal wall)
- 8. **Uncovered string**: in gastroschisis, the herniated abdominal organs are NOT covered by peritoneum
- 9. **Elephant seal**: omphalocele (failure of GI contents to return to the abdominal cavity during embryologic development)
- 10. Elephant seal collar: omphalocele is a true umbilical hernia (through umbilical ring)
- 11. **Hand covered by puppet**: in omphalocele, the herniated abdominal organs ARE covered by peritoneum
- 12. **Trisomy triplets with chromosome waistbands**: omphaloceles are associated with trisomies (e.g. trisomy 18, 13) and other congenital defects (e.g. cardiac abnormalities, neural tube defects)
- 13. **Intestines springing out of box** : congenital diaphragmatic hernia (CDH) (abdominal organs herniate through the left hemidiaphragm and into the thorax)
- 14. **Scrunched up lung shirt**: congenital diaphragmatic hernia can cause pulmonary hypoplasia and pulmonary hypertension (due to compression of the lungs by abdominal organs)
- 15. **Skull and X bones**: chest X-ray can be use to diagnose congenital diaphragmatic hernia (bowel gas and abdominal organs above the diaphragm)
- 16. Broken tracheal and esophageal slide : tracheoesophageal fistula (TEF)
- 17. "SEPTEMBER": TE fistulas are a result of failure of SEPTATION of the tracheoesophageal tube

- 18. "4th and 5th": tracheoesophageal septation normally occurs at 4-5 weeks of gestation
- 19. **Tube-slide connection**: pure TE fistula (trachea and esophagus are connected by a bridge) (rare)
- 20. Stuck in tube: esophageal atresia (normal trachea)
- 21. **Distal tube connection**: type-C TE fistula (most common) (proximal esophagus ends as blind pouch, distal esophagus connects to trachea)
- 22. Vomiting white while stuck on esophageal ring : type-C TE fistula causes non-bilious emesis (proximal esophagus ends in blind pouch \rightarrow food never reaches intestines)
- 23. **Distended belly stuck in tube**: type-C TE fistula causes abdominal distention (due to flow of air from trachea into stomach
- 24. **Refluxing onto lung shirt**: type-C TE fistula causes recurrent pneumonitis and aspiration pneumonia (due to reflux of acidic stomach contents into lungs)
- 25. **Wet baby doll**: type-C TE fistula causes polyhydramnios in utero (due to fetus unable to swallow amniotic fluid)
- 26. **Abnormal doll**: TE fistula can be associated with other congenital anomalies, including VACTERL association)
- 27. "C" curved tube : duodenum
- 28. **Collapsed C tube**: duodenal atresia occurs when second portion of duodenum fails to recanalize
- 29. **Green vomit**: duodenal atresia presents with BILIOUS emesis (due to obstruction distal to Ampulla of Vater)
- 30. **Two fun domes with skull and X bones**: on X-ray, duodenal atresia causes the "double bubble" sign (one bubble of air in stomach and one in proximal duodenum)
- 31. Obstructing end of tube : distal intestinal atresia (jejunum or ileum)
- 32. **Compressing red vascular hair**: distal intestinal atresia is caused by in-utero vascular accident (decreased blood flow to intestine → decreased delivery of nutrients and oxygen → impaired intestinal growth)
- 33. **Green vomit**: distal intestinal atresia presents with bilious emesis (due to obstruction distal to Ampulla of Vater)
- 34. **Distended belly**: distal intestinal atresia presents with abdominal distension (due to accumulation of air proximal to obstruction)

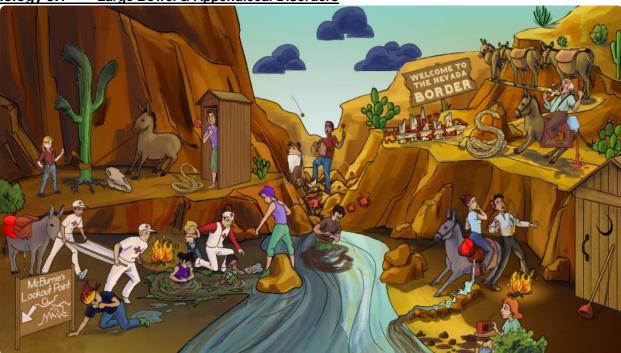


- 35. **Violinist**: vitelline duct (omphalomesenteric duct, connects ileum to yolk sac during fetal life)
- 36. **Meckel hecklers**: Meckel diverticulum (true diverticulum in the ileum) forms if vitelline duct fails to obliterate
- 37. **Gastric violin**: Meckel diverticula can contain gastric mucosa (most common) and other ectopic tissue (pancreatic, endometrial, etc.)
- 38. Tarry black paint : Meckel diverticula can present with painless melena (functional ectopic gastric tissue → ulceration and bleeding)
- 39. **"2" signs**: Meckel diverticulum occurs in 2% of population, exists 2 feet proximal to ileocecal valve, 2 inches in length, presents ~ 2 years of age, has 2:1 male:female ratio
- 40. **Technique book**: Meckel diverticulum can be diagnosed with a technetium-99m pertechnetate scan (gastric mucosa takes up technetium-99m)
- 41. HORSE-SPRING ride: Hirschsprung's disease
- 42. **Migrating CREST border**: Hirschsprung's develops due to failed neural crest cell migration (normally migrate from the embryonic ectoderm into organs)
- 43. **Disconnected plug**: in Hirschsprung's disease, the impaired migration of neural crest cells leads to absent innervation (absent neural ganglia) of the distal colon (almost always including the rectum)
- 44. **Tightly wound rectum spring**: in the absence of innervation, the rectal muscles remain contracted
- 45. **Dilating "S" slinky**: in Hirschsprung's disease, colon proximal to the constriction (usually sigmoid colon) fills with stool and dilates (forming "megacolon")
- 46. **Blackened stool stuck in spring**: Hirschsprung's disease presents with failure to pass meconium within 48 hours after birth

- 47. **Yo-yo not rotating**: intestinal malrotation (failure to undergo normal 270 degree counterclockwise rotation during fetal development)
- 48. **Yo-yo in RUQ pocket**: in intestinal malrotation, the cecum is located in right upper quadrant
- 49. **String compressing belly**: intestinal malrotation can cause duodenal obstruction (due to fibrous bands ["Ladd's bands"] that connect abnormally located cecum to retroperitoneum
- 50. **Distended belly & green face**: in intestinal malrotation, duodenal obstruction can lead to abdominal distension and vomiting
- 51. **Knot in waist sweater**: in intestinal malrotation, twisting around Ladd's bands can cause midgut volvulus (surgical emergency due to risk of bowel necrosis)
- 52. Death metal baby: necrotizing enterocolitis (NEC)
- $53.\,\textbf{Small}$ emaciated baby : necrotizing enterocolitis (NEC) develops in premature and underweight infants
- 54. **Baby bottle**: necrotizing enterocolitis begins when an infant starts feeding (bacteria introduced into the gut overpowers the immature gastrointestinal immune system)
- 55. Inner layer of bacterial wallpaper: in necrotizing enterocolitis, bacteria invade the bowel wall
- 56. **Big belly & red diaper**: necrotizing enterocolitis presents with abdominal distention and bloody stools
- 57. **Black outlines**: necrotizing enterocolitis causes pneumatosis intestinalis (pockets of gas in intestinal wall, causes black outlines of bowel lumen on X-ray)
- 58. **Skull shirt**: necrotizing enterocolitis can cause extensive bowel necrosis and death

GI Pathology 3.1 Large Bowel & Appendiceal Disorders





- 1. Saddle bags: diverticulOSIS (colonic diverticula)
- 2. **False mucusy teeth**: colonic diverticula are false diverticula (herniation of mucosa and submucosa only)(versus herniation of mucosa, submucosa, and muscularis in true diverticula)
- 3. "BORDER": most colonic diverticula form on the mesenteric border
- 4. **"S" snake** : colonic diverticula are most commonly found in the sigmoid colon
- 5. Red vines penetrating fence : vasa recta arteries penetrate the muscularis at the mesenteric border (creates weakening that facilitates diverticulum formation)
- 6. **Plunger**: chronic constipation is a risk factor for diverticulOSIS (straining generates high pressure within colon)
- 7. **Dumping red** : diverticulOSIS causes painless hematochezia (vasa recta injury \rightarrow rupture)
- 8. Fire next to saddle bag: diverticuLITIS (a complication of diverticuLOSIS)
- 9. Leaking saddle bag : diverticuLITIS is caused by perforation of a diverticulum (increased intraluminal pressure or congealed food particles \rightarrow erosion of diverticular wall \rightarrow perforation)
- 10. Flame bandana: diverticuLITIS presents with fever
- 11. Green face: diverticuLITIS presents with nausea/vomiting
- 12. Left lower abdominal fire : diverticuLITIS presents with LLQ pain (due to location in sigmoid colon) $\,$
- 13. **Red inflamed pouch**: diverticuLITIS can cause abscess formation (can cause partial colonic obstruction)
- 14. **Leaking poopy water** : diverticuLITIS can cause fistulas (most commonly colovesical fistula → pneumaturia, pyuria, fecaluria)
- 15. Murky pool: appendicitis
- 16. **Obstructing rock**: in adults, appendicitis is most commonly due to obstruction from an inspissated fecalith (also fibrosis, malignancy, or carcinoid tumor)
- 17. **Soggy tissues**: in children, appendicitis is most commonly due to obstruction from lymphoid tissue hyperplasia (follows infection)
- 18. **Applying pressure**: in appendicitis, obstruction leads to increased intraluminal pressure (obstruction \rightarrow accumulation of mucus \rightarrow distension \rightarrow pressure on appendiceal wall)
- 19. **Squeezing vascular sleeve**: in appendicitis, vessels and lymphatics in the wall of the appendix are compressed (due to increased intraluminal pressure)
- 20. **Skull shirt**: appendicitis can cause ischemia and necrosis (due compression of vessels in the appendiceal wall)
- 21. Fire next to murky pool : appendicitis causes inflammation
- 22. First responders: in appendicitis, histology shows neutrophil infiltration
- 23. Algae plume : in appendicitis, bacterial overgrowth leads to bacterial invasion into the wall
- 24. inflamed red pouch: appendicitis can lead to abscess formation

- 25. Perforating pouch: appendicitis can lead to perforation
- 26. **Clutching mid-abdomen**: appendicitis initially presents with vague periumbilical pain (inflammation of VISCERAL peritoneum innervated by autonomic nervous system afferent fibers (T10 spinal root → pain in T10 dermatome])
- 27. **Right-sided spiky path**: late appendicitis presents with sharp localized RLQ pain (due to inflammation of PARIETAL peritoneum [somatic afferent fibers])
- 28. "McBurnie's Lookout Point": late appendicitis presents with localized RLQ pain at McBurney's point (% between umbilicus and right anterior superior iliac spine)
- 29. Green face: appendicitis presents with nausea/vomiting
- 30. **Flame bandana** : appendicitis presents with fever (usually later in illness course)
- 31. **Relieving defecation**: irritable bowel syndrome (IBS) presents with vague abdominal pain relieved by defecation
- 32. Watery & clumpy dirt: IBS may present with diarrhea and/or constipation (varying consistency)
- ${\bf 33.} \ \textbf{Radio frequency}: {\sf IBS} \ {\sf presents} \ {\sf with} \ {\sf varying} \ {\sf bowel} \ {\sf movement} \ {\sf frequency}$
- 34. **Twisted leash**: large bowel volvulus (twisting of a portion of bowel around its mesentery)
- 35. Cactus roots: in volvulus, the bowel twists at its mesenteric roots
- 36. **Dead cactus**: volvulus can cause strangulation (leads to ischemia and necrosis)
- 37. Right gun pouch: in children, volvulus occurs in the cecum
- 38. "S" snake : in adults, volvulus occurs in the sigmoid
- 39. **Plunger**: chronic constipation is the most common risk factor for large bowel volvulus
- 40. Obstructed canyon: large bowel obstruction
- 41. Cancer crabs : malignancy is the most common cause of large bowel obstruction
- 42. large painful belly: large bowel obstruction presents with abdominal pain and distention
- 43. **Green face dripping brown water**: large bowel obstruction presents with nausea/vomiting (including feculent vomiting)
- 44. Collie : large bowel obstruction presents with colicky abdominal pain
- 45. "Tink": large bowel obstruction presents with high pitched "tinkling" bowel sounds
- 46. **Drumming**: large bowel obstruction causes hyperresonance to percussion (due to gas-filled bowel)
- 47. **Dark clouds with horizontal base**: on X-ray, large bowel obstruction displays dilated loops of bowel (appears as dark pockets of gas) and air-fluid levels (appears as horizontal contrast lines)

GI Pathology 3.2 Crohn's Disease & Ulcerative Colitis





- 1. **Fiery intestines**: inflammatory bowel disease (IBD) (ulcerative colitis and Crohn disease)
- 2. **Young white guy**: IBD is most common in young people with Caucasian and Jewish ancestry
- 3. Fiery hat: IBD presents with fever
- 4. Thin guy: IBD presents with weight loss
- 5. Clutching abdomen: IBD presents with abdominal pain
- 6. Brown puddle: IBD presents with diarrhea
- 7. Union Cannon: ulcerative colitis
- 8. Rusty rectal stool: ulcerative colitis ALWAYS affects the rectum
- 9. **Rust traveling proximally**: ulcerative colitis spreads proximally from rectum (in a CONTINUOUS pattern)
- 10. **Smooth end of cannon**: in ulcerative colitis, the colon has a "lead pipe" appearance on contrast enema (due to loss of normal haustral projections)
- 11. **First responders with perinuclear battle plan**: pANCA (PERInuclear anti-neutrophil cytoplasmic antibodies) are elevated in ulcerative colitis
- 12. **Eroding through 2 layers**: in ulcerative colitis, inflammation affects ONLY the mucosa and submucosa
- 13. **Dirt islands in hole**: in ulcerative colitis, the colon contains "pseudopolyps" (islands of normal mucosa within ulcerated tissue)
- 14. Inflamed cannonball within wall: on histology, ulcerative colitis displays abscesses within colonic crypts
 15. Shrapnel striking left side: ulcerative colitis presents with LEFT-
- 15. **Shrapnel striking left side**: ulcerative colitis presents with LEFT-sided abdominal pain (due to location in rectum and distal colon)
- 16. **Bloody puddle**: ulcerative colitis presents with bloody diarrhea (frequent and small volume due to rectal inflammation)
- 17. **MEGA cannon**: ulcerative colitis can cause toxic megacolon (inflammation extends to colonic smooth muscle → smooth muscle paralysis → severe colonic dilation)
- 18. Exploding cannon: in ulcerative colitis, toxic megacolon can lead to perforation
- 19. Cancer crab belt: ulcerative colitis increases the risk of colon cancer (bigger risk factor than Crohn disease)
- 20. **Scaly snake in biliary tree**: ulcerative colitis increases the risk of primary sclerosing cholangitis (PSC)(autoimmune disorder of intra- and extrahepatic bile ducts)
- 21. **Cobblestone patches**: Crohn disease occurs as multiple separate areas of disease ("skip lesions")

- 22. Cobblestone path from mouth to anus arch: Crohn disease can affect ANY portion of the GI tract (from mouth to anus)
- 23. **Antibody sac**: anti-Saccharomyces cerevisiae antibodies are often present in ulcerative colitis
- 24. **Hit on right waist**: Crohn disease presents with RLQ abdominal pain (due to location in ileum and cecum)
- 25. **Debris-littered brown puddle**: Crohn disease presents with malabsorption of fats and fat-soluble vitamins (inflammation of distal ileum → poor reabsorption of bile acids → impaired absorption of fat)
- 26. **MacroCAGES**: Crohn disease causes formation of noncaseating granulomas (containing aggregations of macrophages)
- 27. **Broken through all wall layers**: Crohn disease causes TRANSMURAL bowel inflammation
- 28. **Tight abdominal gauze**: Crohn disease can cause fibrotic strictures (transmural inflammation → bowel wall thickening → narrowed lumen)
- 29. **Pulling strings**: in Crohn disease, X-ray shows "string sign" (due to strictures)
- 30. **Obstructing rocks**: in Crohn disease, strictures can cause bowel obstruction
- 31. **Fistulizing trench**: Crohn disease can lead to fistulas (transmural inflammation leads to epithelization across bowel wall \rightarrow connections between bowel and bladder, skin, vagina, or other bowel segments)
- 32. Pale erosions on mouth arch : Crohn disease causes aphthous ulcers in the mouth and esophagus
- 33. Wrapped joints: inflammatory bowel disease can cause arthritis (migratory polyarthritis, sacroiliitis, ankylosing spondylitis)(often HLA-B27)
- 34. **Red spots on shin columns**: inflammatory bowel disease can cause erythema nodosum (painful erythematous nodular rash on shins)(due to inflammation of fat cells)
- 35. **Skin wounds**: inflammatory bowel disease can cause pyoderma gangrenosum (skin ulcers due to neutrophil dysfunction) (not infectious or gangrenous)
- 36. **Shrapnel on kidney tray**: inflammatory bowel disease can cause renal calculi (due to increased absorption of oxalate)
- 37. **Red-rimmed goggles**: inflammatory bowel disease can cause eye disease (anterior uveitis, iritis, episcleritis)(presents with eye pain and redness)
- 38. **5-pointed star of USA**: inflammatory bowel disease is treated with 5-ASA (sulfasalazine, mesalamine)

GI Pathology 3.3 Colorectal Polyps & Cancer



- Mushroom caps: polyps are outgrowths of mucosa most commonly found in the colon (may be pedunculated or sessile) (may be neoplastic or nonneoplastic)
- 2. Hamartomatous hammer: HAMARTOMATOUS polyps (non-neoplastic)
- 3. **Disorganized welcome sign**: HAMARTOMATOUS polyps are disorganized growths of mature cells)
- 4. Happy old guy: HAMARTOMATOUS polyps are MATURE and BENIGN
- 5. **Peutz-pop**: Peutz-Jeghers syndrome presents with hamartomatous polyps in the GI tract (most common in duodenum, can also occur in colon)
- 6. **Domino**: Peutz-Jeghers and juvenile polyposis syndromes (familial causes of HAMARTOMATOUS polyps) are autosomal dominant
- 7. **Pigmented juice spots**: Peutz-Jeghers syndrome presents with pigmented macules around mouth, lips, palms, and genitalia (mucocutaneous hyperpigmentation)
- 8. **Infant with hammer**: JUVENILE POLYPOSIS syndrome (most common in children < 5 years of age) presents with HAMARTOMATOUS polyps
- 9. **Mushroom emerging from the behind**: JUVENILE polyps are most common in the RECTUM and may prolapse through the anal sphincter
- 10. **Bleeding mushroom**: JUVENILE polyps present with lower GI bleeding (hematochezia)
- 11. Little mushroom spots: JUVENILE polyps display small cystic spaces (glands filled with mucin and debris)
- 12. Creeping crab beetle: JUVENILE POLYPOSIS syndrome is associated with an increased risk of colon cancer (despite polyps being benign)
- 13. **Big hyperplastic mushroom**: HYPERPLASTIC polyp (non-neoplastic epithelial proliferation)
- 14. Happy lilipolyps: HYPERPLASTIC polyps are BENIGN
- 15. Mucous-y goblet shrooms : HYPERPLASTIC polyps are epithelial proliferations that contain abundant goblet cells
- 16. **Sawing near the surface**: HYPERPLASTIC polyps have serrated architecture only at the SURFACE (crypts retain smooth shape)
- 17. Sawing on stalk-less mushroom cap: SESSILE SERRATED polyp (premalignant flat lesion of the colon)
- 18. Sawing all the way through: SESSILE SERRATED polyps have serrated architecture THROUGHOUT (crypts included)
- 19. Creeping crab beetle: SERRATED POLYPS & ADENOMAS have the potential to transform into cancer
- 20. **ADDING** shingles to mushroom house: ADENOMATOUS polyps (ADENOMAS) are the most common type of colon polyp
- 21. Creeping crab beetle: SERRATED POLYPS & ADENOMAS have the potential to transform into cancer

- 22. Oversized mushroom next to crab beetle: larger ADENOMAS are more likely to transform into cancer
- 23. "1" horn: ADENOMAS > 1 cm are significantly more likely to transform into cancer)
- 24. Increasingly disorganized purple weeds next to crab beetle :
- ADENOMAS with a greater degree of dysplasia (more hyperchromatic, elongated, and stratified epithelium) are more likely to transform into cancer
- 25. **TUBULAR horn**: TUBULAR ADENOMAS (small pedunculated polyps composed of dysplastic mucosal cells that form tube-shaped glands)
- 26. **Tubular perforations**: TUBULAR ADENOMAS display round tubular gland cross sections on histology
- 27. Finger-like stalk-less fungus: VILLOUS ADENOMAS (sessile, larger than tubular adenomas, long glands with villi-like projections)
- 28. Villus fungus next to crab beetle: VILLOUS ADENOMAS are more likely to transform into cancer (compared to TUBULAR ADENOMAS)
- 29. **Polyp family**: Family adenomatous polyposis (FAP) (inherited syndrome that causes thousands of adenomatous polyps early in life)
- 30. Domino: FAP is autosomal DOMINANT
- 31. "A Pulled Carrot": in germline mutation in the APC gene causes FAP)
- 32. Cancer-fighting ribbon : APC is a tumor suppressor gene
- 33. A single pulled carrot stalk: in FAP, only one APC allele has a germline mutation (the second mutation occurs during person's lifetime) ("second hit")
- 34. **Abundant mushrooms**: FAP can present with thousands of polyps in the colon (as visualized by colonoscopy)
- 35. Family crab beetle: FAP progresses to colon cancer in nearly 100% of cases (treatment is prophylactic colectomy)
- 36. **Gardner within mushroom field**: GARDNER syndrome (FAP variant) (inherited disorder presenting with adenomatous polyps and SOFT TISSUE TUMORS)
- 37. **Bone garden fence**: GARDNER syndrome presents with osteomas (benign bone tumor)
- 38. **Skin rash on gardner**: GARDNER syndrome presents with cutaneous lesions
- 39. **Overgrown sunglasses on gardner**: GARDNER syndrome presents with hypertrophy of retinal pigment epithelium
- 40. **Tooth gardening stool**: GARDNER syndrome presents with dental abnormalities (impacted and supernumerary teeth)
- 41. Elder lilipolyp wearing turban: TURCOT syndrome (FAP variant) presents with colon adenomas and BRAIN tumors (medulloblastomas)

GI Pathology 3.3 Colorectal Polyps & Cancer



- 42. **Crab beetle attacking Lilipolyp Village**: colorectal cancer (most commonly adenocarcinoma 3rd most common cancer and 2nd most common cause of cancer death)
- 43. **Old man lilipolyp riding crab beetle**: peak incidence of colorectal cancer is 60-70 years of age, with men affected slightly more than women
- 44. **Family photo**: family history is a risk factor for colorectal cancer (even in the absence of a known familial syndrome)
- 45. Smoker: smoking is a risk factor for colorectal cancer
- 46. Alcohol bottle: alcohol use is a risk factor for colorectal cancer
- 47. Round lilipolyp: obesity is a risk factor for colorectal cancer
- 48. Fiery intestines : inflammatory bowel disease is a risk factor for colorectal cancer
- 49. "A Pulled Carrot": mutations in the APC gene lead to the initial growth of small ADENOMATOUS POLYPS (ADENOMAS)
- 50. **Carrots**: the APC gene product controls the concentrations of beta-CATENIN and interacts with E-CADHERIN
- 51. Cancer-fighting ribbon: APC is a tumor suppressor gene
- 52. **Cancerous rat**: mutations in the K-RAS proto-oncogene leads to unstoppable cell division (K-RAS encodes for a protein that regulates the cell cycle)
- 53. **Mushrooms increasing in size**: ADENOMAS increase in size as a result of mutations in K-RAS
- 54. **Broken city wall**: basement membrane invasion marks malignant transformation of an ADENOMA into an ADENOCARCINOMA
- 55. **Broken checkpoint**: mutations in the p53 gene contribute to malignant transformation (a tumor suppressor) (normally prevents G1 to S phase transition)
- 56. **Broken "Don't CCross"**: deletions in the Deleted in Colon Cancer (DCC) gene contribute to malignant transformation (tumor suppressor) (normally activates apoptosis pathway)
- 57. **Cauliflower on the RIGHT**: RIGHT-sided colon cancer (proximal ascending colon) presents as an exophytic mass (usually NON-obstructive despite extension into the lumen)
- 58. Occult watchman dropping iron tools: RIGHT-sided colon cancer often presents with iron deficiency anemia (from occult GI bleeding)
- 59. "Path Obstructed" on LEFT: LEFT-sided colon cancer (descending colon) may present with colonic obstruction (due to luminal narrowing from infiltrative ulcerative lesions) leading to changes in bowel habits

- 60. **Apple core on left**: LEFT-sided colon cancer can present with an "apple core" lesion on imaging (due to luminal narrowing)
- 61. Sitting in red puddle: LEFT-sided colon cancer presents with hematochezia
- 62. **Periscope**: colonoscopy is the gold standard for screening and diagnosis of colon cancer (early detection and removal of ADENOMAS can prevent cancer)
- 63. Fibrous hat : a high-fiber diet decreases the risk of colon cancer
- 64. Aspirin umpire guard : regular aspirin use decreases the risk of colon cancer
- 65. "CEA": carcinoembryonic antigen (CEA) can be used to monitor progression or recurrence of colon cancer (too nonspecific for cancer diagnosis)
- 66. **Lynch inchworm**: Lynch syndrome (hereditary non-polyposis colorectal cancer [HNPCC]) is the most common familial cause of colorectal cancer (NO adenomatous polyps)
- 67. Domino bridge: Lynch syndrome is autosomal DOMINANT
- 68. **Mismatched colors**: Lynch syndrome is caused by germline mutations in mismatch repair genes (MSH2 and MLH1)
- 69. Cancer fighting ribbon : mismatch repair genes (MSH2, MLH1) are tumor suppressor genes
- 70. **One falling antennae**: Lynch syndrome is caused by inheritance of one germline mutation (second mutation is acquired during person's lifetime)
- 71. **Crab belt buckle**: Lynch syndrome is also associated with increased risk of other abdominal/pelvic cancers (endometrial, ovarian, stomach, small bowel, hepatobiliary)

GI Pathology 3.4 Anorectal Disorders





- 1. Canal entrance : the rectum
- 2. Canal exit: the anus
- 3. Tooth bridge: the rectum and anus converge at the dentate (pectinate) line
- 4. **Columnar bricks**: SUPERIOR to the dentate line, tissue derived from the endoderm (mucosa is COLUMNAR)
- 5. **Squamous tile**: INFERIOR to the dentate line, tissue derived from the ectoderm (mucosa is SQUAMOUS)
- 6. Rope plexus under stomach tower: SUPERIOR to the dentate line, innervation is via the inferior hypogastric plexus
- 7. "Automatic Sensor": SUPERIOR to the dentate line, innervation is by AUTONOMIC nerves (e.g. inferior hypogastric plexus) which detects STRETCH only
- 8. **Pedaling**: INFERIOR to the dentate line, innervation is via the inferior rectal nerve (a branch of the pudendal)
- 9. **Pedaler in pain**: any pathology BELOW the dentate line can cause PAIN (innervation via SOMATIC nerves)
- 10. **Schooner's PORThole**: ABOVE the dentate line, venous blood drains into the PORTAL circulation
- 11. "International Marine Vessel" : SUPERIOR to the dentate line, venous blood travels via the superior rectal vein → Inferior Mesenteric Vein → portal vein
- 12. **Pudendal paddle boat**: BELOW the dentate line, venous blood drains into the SYSTEMIC circulation (inferior rectal vein → internal pudendal vein → internal iliac vein)
- 13. **Dilated arteriovenous pipes**: hemorrhoids are dilated vascular structures in the submucosa of the anal canal
- 14. Dilated upper pipes: INTERNAL hemorrhoids occur ABOVE the dentate line
- 15. **Bleeding dilated pipes**: INTERNAL hemorrhoids cause bleeding (blood coating stool or dripping after defecation)
- 16. Happy canal worker: INTERNAL hemorrhoids are painless (due to autonomic innervation)
- 17. Dilated lower pipes: EXTERNAL hemorrhoids occur INFERIOR to the dentate line
- 18. **Thrombotic driftwood**: thrombosis of EXTERNAL hemorrhoids can cause acute pain
- 19. **Older man using plunger**: hemorrhoids are associated with older adults and chronic constipation (e.g. opioid use, due to straining with defecation)
- 20. Pregnant woman : hemorrhoids occur in pregnancy (increased intra-abdominal pressure and IVC compression → venous pooling and engorgement)
- 21. **Connected pipes**: the upper anus/lower rectum is a portocaval junction (the PORTAL venous system communicates with the SYSTEMIC venous system)
- 22. Steaming PORThole : high pressure in the PORTAL venous system (portal hypertension) can cause hemorrhoids (pressure transmitted to SYSTEMIC venous system)
- 23. Barge pushing through anal gate : in rectal prolapse, the rectum pushes through a weak pelvic floor \rightarrow prolapse through the anus
- 24. Telescoping: in rectal prolapse, the layers of mucosa fold on each other

- 25. Plungers: chronic constipation can cause rectal prolapse
- 26. **Old woman holding baby**: rectal prolapse occurs in elderly women with a weak pelvic floor (history of multiple pregnancies)
- 27. **Kid coughing** : whooping cough can cause rectal prolapse (coughing \rightarrow increased intraabdominal pressure)
- 28. **Thick tree sap**: cystic fibrosis can cause rectal prolapse (due to coughing and strained defecation)
- 29. **Top of tree cleft**: pilonidal abscesses form at the natal cleft (located at the top of the gluteal cleft)
- 30. Hairy tree bulge: breakdown of hair follicles in natal cleft \rightarrow pit collecting debris and flora \rightarrow infection \rightarrow pilonidal abscess
- 31. Machete draining \mbox{pus} : a pilonidal abscess is a painful pustulent sacrococcygeal mass
- 32. Inner tube dragged over puddle with sharp rocks: anal fissures are initiated by trauma to the anal sphincter (e.g. hard stool, severe diarrhea, vaginal delivery)
- 33. **Shaky lines**: in anal fissures, stretching and tearing of anal sphincter \rightarrow spastic response \rightarrow worsening stretching and tearing
- 34. Posterior inner tube rider : the majority of anal fissures are POSTERIOR near the midline
- 35. Hat flaps : many fissures have an anal tag
- 36. Anterior cobble stones with skip lesions: ANTERIOR anal fissures are associated with Crohn's disease, malignancy, and infection
- 37. Pipe draining to ocean: an orectal fistulas run from the anus or rectum to the skin surface
- ${\it 38.} \ \textbf{Epithelial pipe}: fistulas \ are \ tunnels \ that \ eventually \ become \ epithelialized)$
- 39. Radiation sign: pelvic radiation can cause anorectal fistulas
- 40. **Skipping cobble stones** : Crohn's disease can cause anorectal fistulas (transmural inflammation of bowel \rightarrow sinus tracts \rightarrow fistulas
- 41. **Hermit crab shell** : squamous cell carcinoma is the most common anal cancer and displays keratinization
- 42. **Un-shelled crab**: basaloid carcinoma (nonkeratinized squamous cell) of the anus arises near the dentate line)
- 43. Crab with glandular sponge: rectal adenocarcinoma arises above the dentate line and behaves like colon adenocarcinoma
- 44. Pill-bugs : infection with high risk HPV (16, 18) is a risk factor for squamous cell carcinoma of the anus
- 45. White wizard hat: HIV infection is a risk factor for squamous cell carcinoma of the anus
- 46. **Crutches**: immunocompromised states are a risk factor for squamous cell carcinoma of the anus
- 47. Smoker: smoking is a risk factor for squamous cell carcinoma of the anus
- 48. Kissing men; men who have sex with men (MSM) are at higher risk for squamous cell carcinoma of the anus :
- 49. Pooling blood : anorectal cancer often presents with bleeding

Hepatobiliary 1.1 - Lab Evaluation of Liver Injury & Hyperbilirubinemia





- 1. **Iron wheel hub**: bilirubin is produced by the breakdown of heme (iron + porphyrin ring)
- 2. Spleen sac : red blood cells are broken down by splenic macrophages
- 3. **MacroCAGE pen**: within splenic macrophages, heme is broken-down into iron (gets recycled) and the porphyrin ring
- 4. Uncollared billy goat : the porphyrin ring is converted into unconjugated bilirubin
- 5. Uncollared billy goat with photo albumin: in blood, unconjugated bilirubin is bound to albumin (to increase solubility)
- 6. **Goat tied to Goat Ride sign**: phase II conjugation to GLUCURONIDE by UDP-glucuronyltransferase converts unconjugated bilirubin to conjugated bilirubin
- 7. Collared goat drinking water: conjugated bilirubin is water soluble (excreted into bile)
- 8. Baby goat in urine puddle next to pipe: conjugated bilirubin is broken down into urobilinogen by intestinal flora
- 9. Baby goat stuck in mud next to pipe: some of the urobilinogen in the intestine is oxidized into stercobilin, which give stool its brown color
- 10. Liver spot on urine goat : some of the urobilinogen formed in the intestine is reabsorbed into the bloodstream and returned to the liver
- 11. **Goat in urine puddle**: some urobilinogen is excreted in the urine; it may also be oxidized to urobilin, which gives urine its yellow color
- 12. **UNcollared goat pen**: UNconjugated hyperbilirubinemia (direct/total bilirubin fraction < 20%)
- 13. Fence between goat pens: mixed hyperbilirubinemia (direct/total bilirubin fraction between 20-50%)
- 14. **Collared goat pen**: conjugated hyperbilirubinemia (direct/total bilirubin fraction >50%)
- 15. **Tomatoes on ground**: hemolytic anemia increases heme breakdown, which increases unconjugated bilirubin
- 16. **Uncollared goat kicking tomatoes**: hemolysis (e.g. hemoglobinopathy, RBC structural defect, enzyme defect) increases the breakdown of heme, leading to elevated unconjugated bilirubin
- 17. Baby urine goat kicking tomatoes: increased metabolism of bilirubin with hemolysis also leads to increased levels of urobilinogen in the urine
- 18. Slow Farmer Gilbert with leash: in Gilbert syndrome, decreased activity of UDP-glucuronyltransferase leads to increased unconjugated bilirubin
- 19. **Kid hiding behind mom**: Gilbert syndrome (and Crigler-Najjar) is an autosomal recessive disorder
- 20. **Stressed yellow kid**: Gilbert syndrome is a benign condition and leads to mild jaundice in times of physiological stress
- 21. Catching cricket in a jar : Crigler-Najjar is an autosomal recessive lack of UDP-glucuronyltransferase → unconjugated hyperbilirubinemia
- 22. **Uncollared goat jumping over cricket**: complete lack of UDP-glucuronyltransferase (in Crigler-Najjar) leads to dramatically elevated unconjugated bilirubin in infants
- 23. **Baby hitting head on ground**: unconjugated bilirubin deposition in the brain can cause encephalopathy, brain damage, and death in infants with Crigler-Najjar
- 24. **Corn on fence**: irreversible neurologic dysfunction caused by bilirubin deposition in the brain is known as kernicterus (seen in Crigler-Najjar)

- 25. **Baby in yellow**: slow activity of UDP-glucuronyltransferase in newborns leads to physiologic jaundice of the newborn (unconjugated hyperbilirubinemia)
- 26. **Blue bug zapper**: treatment of physiologic jaundice of the newborn usually only requires phototherapy (converts trans-bilirubin to water soluble cis-bilirubin and aids excretion)
- 27. **Yellow baby on breast**: breast FEEDING jaundice (lack of enteral feeding \rightarrow slow bile excretion \rightarrow increased unconjugated bilirubin and jaundice in 1st week) and breast MILK jaundice (substance in milk inhibits UDP-glucuronyltransferase \rightarrow increased unconjugated bilirubin and jaundice in 2nd week)
- 28. XXX moonshine jug on fence : alcoholic hepatitis is one of the leading causes of mixed hyperbilirubinemia
- 29. **Hippo truck above fence**: viral hepatitis is one of the leading causes of mixed hyperbilirubinemia
- 30. **Spraying collared goat**: hepatocyte damage in hepatitis releases CONJUGATED bilirubin into the serum → excreted in the URINE (always abnormal)
- 31. **ToASTing with alcohol** : in alcoholic hepatitis, both AST and ALT are elevated (with AST:ALT ratio >2)
- 32. ${\bf GadGeT\ knife}$: alcohol induces microsomal enzyme activity, leading to elevated GGT
- 33. **Elevated MALT milkshake**: in viral hepatitis, both AST and ALT are elevated (much greater elevation in ALT)
- 34. **Pellets clogging green pipe**: blockage of bile outflow (e.g. choledocholithiasis) leads to cholestasis and elevated conjugated bilirubin
- 35. Collared goat in yellow trough: in cholestasis, conjugated bilirubin leaks into the serum and is excreted in the urine (always abnormal)
- 36. Dark yellow water: in cholestasis, urine is dark in color due to elevated levels of conjugated bilirubin
- 37. Whitish poops under trough : blockage of bile outflow leads to reduced urobilinogen and stercobilin in the intestine \rightarrow pale stool
- 38. ChALK and GadGeT knife: diseases that causes cholestasis lead to biliary damage, increasing alkaline phosphatase and GGT
- 39. Yellow collared goat : conjugated hyperbilirubinemia (e.g. cholestasis) can cause jaundice
- 40. **Garbage BIN blocking exit**: Dubin-Johnson syndrome (mutation of intrahepatic bile duct membrane proteins → decreased bilirubin excretion and conjugated hyperbilirubinemia)
- 41. Hiding behind bin : Dubin-Johnson syndrome is autosomal recessive
- 42. **Black trash bags in bin**: black pigment collects in hepatocyte lysosomes (in Dubin-Johnson syndrome)]
- 43. **Liver-shaped trash bag**: black pigment collecting in hepatocyte lysosomes gives the liver a black appearance (in Dubin-Johnson syndrome)
- 44. **Rotating windmill**: Rotor syndrome (defect in bile excretion similar to Dubin-Johnson syndrome causes conjugated hyperbilirubinemia (without black liver)
- 45. Hiding behind windmill: Rotor syndrome is autosomal recessive
- 46. **Kid holding fibrosed green twig**: biliary atresia is caused by fibrosis of the bile ducts, leading to cholestasis and conjugated hyperbilirubinemia
- 47. **Yellow onesie**: cholestasis in biliary atresia leads to signs of cholestasis (jaundice, dark urine/light stool) and cholestatic labs (conjugated hyperbilirubinemia, transaminitis, elevated alk phos and GGT)

Hepatobiliary 1.2 - Gallbladder & Biliary Tract Disease



- 1. Green outpouching: the gallbladder
- 2. Short path connecting to larger bocce court : the cystic duct connects the gallbladder to the hepatic duct \to forms the common bile duct
- 3. Bocce ball court : common bile duct
- 4. **Sea-GALL stones**: gallstones are solidifications of liquid bile that develop in the gallbladder
- 5. Yellow bocce ball: cholesterol stones appear yellow
- 6. Black bocce ball with collared billy goat : black pigmented stones contain conjugated bilirubin
- 7. **Cholesterol noodles**: cholesterol (stored and processed in the liver, and circulates as part of lipoproteins)
- 8. **SALT**: some cholesterol is excreted in the GI tract in the form of bile salts (aid in fat digestion)
- 9. " α " with "7" handle : 7- α -hydroxylase is the rate limiting enzyme in the conversion of cholesterol into bile salts
- 10. **Cholesterol noodles on green table**: bile contains free cholesterol (which is fat-soluble)
- 11. Salt on green table: bile contains bile salts (amphipathic and water soluble, helps make bile more water soluble)
- 12. **Squiggly phospholipid fork**: bile contains phosphatidylcholines (lecithins), which help make bile more water soluble
- 13. **Precipitating cholesterol noodles**: excessive cholesterol relative to amount of bile salts → supersaturated cholesterol
- 14. **Yellow bocce ball** : supersaturated cholesterol \rightarrow formation of cholesterol stones (yellow)
- 15. **Overweight, older woman with baby**: risk factors for cholesterol gallstones include "fat, fertile, forty, female"
- 16. **Estrogen earrings**: high estrogen states are responsible for many of the cholesterol gallstone risk factors ("fat, fertile, forty, female")
- 17. **Woman asking for more noodles**: estrogen increases the biosynthesis of cholesterol by upregulating HMG-CoA reductase activity
- 18. "Horatio, Mario, Gio & Co Ristorante" sign: HMG CoA reductase (the rate determining enzyme in the pathway responsible for biosynthesis of cholesterol in the liver)
- 19. **Deflated tires on green vespa** : DECREASED gallbladder motility \rightarrow bile stasis \rightarrow gallstone formation

- 20. **Vespa leaking fluid** : gallbladder reabsorbs water from bile (gallbladder stasis leads to precipitation of cholesterol \rightarrow cholesterol gallstones)
- 21. **Pregnant woman**: during pregnancy, elevated progesterone levels decrease gallbladder motility → increased reabsorption of water from bile → cholesterol gallstones
- 22. **STOP sign** : somatostatin administration decreases gallbladder motility \rightarrow increased reabsorption of water from bile \rightarrow cholesterol gallstones
- 23. **TPN bag** : administration of total parenteral nutrition decreases gallbladder motility \rightarrow increased reabsorption of water from bile \rightarrow cholesterol gallstones
- 24. "FAST" : fasting causes decreased gallbladder motility \rightarrow increased reabsorption of water from bile \rightarrow cholesterol gallstones
- 25. Lysing tomatoes with black ball: black pigmented stones are composed of conjugated bilirubin due to intravascular hemolysis (e.g. hemoglobinopathies, G6PD deficiency, hereditary spherocytosis)
- 26. **Brown bocce ball with uncollared billy goat**: brown pigmented stones contain unconjugated bilirubin
- 27. **Brown bocce ball next to candle**: brown pigmented stones are associated with biliary tree infections (bacteria deconjugate bilirubin)
- 28. **Cobblestone skip lesions**: Crohn's disease increases the risk of gallstones (usually pigmented stones due to increased levels of bilirubin in bile)
- 29. **Pulled from fatty meal**: gallstones can cause waxing and waning right upper quadrant or epigastric pain (biliary colic), worse after a large or fatty meal
- 30. Collie contracting leash : fatty acids reach duodenum \rightarrow release of CCK \rightarrow gallbladder contracts against a stone INTERMITTENTLY obstructing gallbladder outlet \rightarrow biliary colic)
- 31. **Collie retrieving stone**: gallstones present with BILIARY COLIC (due to gallbladder contracting against a stone INTERMITTENTLY obstructing gallbladder outlet)
- 32. Nauseated : BILIARY COLIC can be associated with nausea and vomiting

Hepatobiliary 1.2 - Gallbladder & Biliary Tract Disease



- 33. **Bocce ball causing green table fire**: ACUTE CHOLECYSTITIS is inflammation of the gallbladder due to a stone PERSISTENTLY obstructing gallbladder outlet)
- 34. Wax accumulating behind bocce ball : in cholecystitis, mucus accumulates behind impacted stone
- 35. Fire accumulating behind bocce ball : phospholipases hydrolyze biliary enzymes --> release of caustic substances \rightarrow mucosal inflammation
- 36. Extending arms to exert pressure : worsening inflammation \rightarrow gallbladder distension and increased intraluminal pressure (in ACUTE CHOLECYSTITIS)
- 37. Clamping down on red shirt sleeve : increased intraluminal pressure \rightarrow compression of vasculature (in ACUTE CYSTITIS)
- 38. Torn necrotic skull shirt : compression of vasculature \rightarrow gallbladder ischemia \rightarrow necrosis and perforation (in ACUTE CYSTITIS)
- 39. **E. COLA**: in ACUTE CYSTITIS, bacterial overgrowth (most commonly E. coli) can occur inside the obstructed gallbladder \rightarrow peritonitis if perforation occurs
- 40. **Painful gasping**: ACUTE CHOLECYSTITIS can present with Murphy's sign (pain on palpation of RUQ during deep inspiration)
- 41. Flame bandana: ACUTE CHOLECYSTITIS can present with fever and leukocytosis
- 42. **Bullhorn with thick outer rim**: on ultrasound, ACUTE CHOLECYSTITIS demonstrates a thickened gallbladder wall (sometimes gallstones are visible)
- 43. **Hiding from the bocce ball**: radioscintigraphy (aka HIDA scan) can be used to diagnose an impacted stone causing cholecystitis (radiotracer does not enter the gallbladder)
- 44. **Sick guy knocking over candles**: critically ill patients may present with ACALCULOUS CHOLECYSTITIS (caused by bile stasis, not obstructive gallstones)
- 45. Grandfather clock : chronic cholecystitis
- 46. **Multiple inflammatory candles**: CHRONIC CHOLECYSTITIS is caused by repeated episodes of acute cholecystitis (symptoms are similar to acute cholecystitis)
- 47. **Herniating wax**: CHRONIC CHOLECYSTITIS is diagnosed by histology (herniation of gallbladder mucosa into muscular wall [Rokitansky-Aschoff sinus])
- 48. **Bocce ball further down court**: CHOLEDOCHOLITHIASIS (obstruction of the common bile duct by gallstones)

- 49. **Alk phos chalk**: in CHOLESTASIS due to choledocolithiasis, alkaline phosphatase is elevated (also produced by bone and placental tissue)
- 50. **GGT gadget** : in CHOLESTASIS due to choledocolithiasis, gamma glutamyl transpeptidase (GGT) is elevated (specific to bile duct)
- 51. **Collared billy goat**: in CHOLESTASIS due to choledocolithiasis, conjugated bilirubin is elevated (DIRECT hyperbilirubinemia)
- 52. **Bocce ball in ampullary pot**: gallstones at the ampulla of Vater can obstruct the common bile duct and the pancreatic duct)
- 53. **Pancreas sponge**: gallstones are the most common cause of acute pancreatitis (due to obstruction at the Ampulla of Vater)
- 54. Fire ascending bocce court : ascending CHOLANGITIS (acute inflammation of the bile ducts)
- 55. **Obstructing bocce ball**: ascending CHOLANGITIS is usually caused by obstruction (such as by gallstones)
- 56. **E. COLA bottle**: ascending CHOLANGITIS is caused by migrating bacteria from the small intestine (e.g E. coli, Enterococci, Clostridium, Bacteroides)
- 57. **Charcot short coat**: CHOLANGITIS presents with Charcot's Triad (jaundice, fever, right upper quadrant pain)
- 58. Yellow pantsuit: CHOLANGITIS presents with jaundice
- 59. Flaming red hair: CHOLANGITIS presents with fever
- 60. Hit in the RUQ: CHOLANGITIS presents with RUQ pain
- 61. **Bocce ball eroding out of bocce court**: gallstones can erode through biliary wall → creates a fistula between biliary tract and adjacent small bowel (biliary-enteric fistula)
- 62. "Path Obstructed": a biliary-enteric fistula may lead to GALLSTONE ILEUS (obstruction of the ileum by a gallstone)
- 63. Aerated bottle of sparking water: air in the biliary tree is a sign of biliary-enteric fistula (and possible GALLSTONE ILEUS)
- 64. **Crab on chronic table**: gallbladder adenocarcinoma is the most common gallbladder cancer (chronic gallstones and cholecystitis are risk factors)
- 65. **Spilled milk** : chronic gallbladder inflammation (such as from chronic cholecystitis) causes dystrophic calcification \rightarrow can progress to carcinoma
- 66. **Porcelain vase**: dystrophic calcification of the gallbladder causes "porcelain gallbladder" appearance on X-ray
- 67. **Cauliflower** : gallbladder adenocarcinoma can form an exophytic ("cauliflower-like") mass



- 1. **Green biliary vine INSIDE teepee** : primary biliary cholangitis (PBC) is autoimmune-mediated destruction of INTRAhepatic bile ducts
- 2. **Broken mitochondrial shield**: PBC is an autoimmune disorder resulting from genetic and environmental injury to biliary epithelial mitochondria
- 3. Arrows next to mitochondrial shield: PBC involves development of antimitochondrial antibodies (AMA)
- 4. Female hunter: PBC primarily affects middle-aged women
- 5. **Autoimmune arrows**: PBC often develops in patients with pre-existing autoimmune disease (e.g. thyroid disease, scleroderma, Sjogren's)
- 6. Macro-CAGES: in PBC, granulomas form within the portal triads
- 7. **ChALK**: alkaline phosphatase is elevated with cholestasis (PBC, PSC, cholangiocarcinoma)
- 8. **Gadget knife**: gamma glutamyl transpeptidase (GGT) is elevated with cholestasis (PBC, PSC, cholangiocarcinoma)
- 9. **Collared billy goat** : cholestasis causes CONJUGATED (DIRECT) hyperbilirubinemia (PBC, PSC, cholangiocarcinoma)
- 10. **Antibody dream catchers** : in PBC, levels of immune proteins (IgM, etc.) are elevated
- 11. **Sleepy goat**: cholestasis presents with fatigue (most common presenting symptom) (PBC, PSC, cholangiocarcinoma)
- 12. **Black facepaint**: PBC can cause hyperpigmentation (due to excess melanin deposition in the skin)
- 13. **Collections of yellow feathers**: PBC can cause xanthomas (including eyelid xanthelasmas)(due to hypercholesterolemia from cholestasis)
- 14. **Itchy face chalk**: cholestasis can cause pruritus (possibly secondary to bile acid accumulation) (PBC, PSC, cholangiocarcinoma)
- 15. **Yellow goat**: in advanced disease, cholestasis can cause jaundice (conjugated hyperbilirubinemia) (PBC, PSC, cholangiocarcinoma)
- 16. Liver rock: PBC can progress to cirrhosis
- 17. Cancer crab: PBC increases the risk of hepatocellular carcinoma (via progression to cirrhosis)
- 18. **Chunky muddy water**: PBC can lead to steatorrhea (due to poor absorption of fats from to cholestasis) and malabsorption
- 19. **Broken ADEK dock**: PBC can lead to poor absorption of fat-soluble vitamins (A,D,E,K)(due to cholestasis)
- 20. URSA MAJOR: PBC can be treated with ursodeoxycholic acid]
- 21. **Man at entrance**: primary sclerosing cholangitis (PSC) is autoimmune destruction of bile ducts (primarily occurs in middle-aged men)
- 22. Green biliary vine INSIDE and OUTSIDE teepee: PSC affects INTRAand EXTRAhepatic bile ducts
- 23. Sclerotic snake skins: PSC causes sclerosis of bile ducts

- 24. Antibody arrows: PSC is an autoimmune disorder
- 25. PRESENTING 2 rabbits with "D" bow: PSC is associated with HLA-DR genes (encodes for MHC class II on antigen presenting cells)
- 26. **First responders**: PSC is associated with p-ANCA (perinuclear antineutrophil cytoplasmic antibodies)
- 27. **Antibody arrows SURROUNDING deer**: p-ANCA are ANCA with perinuclear localization (i.e. SURROUNDING the perimeter of the nucleus)
- 28. **Intestinal fire**: PSC is associated with inflammatory bowel disease (usually ulcerative colitis)
- 29. **Onion layers** : on histology, small ducts show layers of concentric scar tissue ("onion skin") \rightarrow destruction
- 30. **Green beads**: imaging in PSC shows "beading" in the biliary tree due to alternating areas of stricture and dilation
- 31. Antibody dream catchers : in PSC, levels of immune proteins (IgM, etc.) are elevated
- 32. **Torch ascending biliary tree**: PSC can present with ascending cholangitis (due to transient blockage of extrahepatic biliary tree)
- 33. Cancer crabs on biliary tree : PSC is a risk factor for cholangiocarcinoma (due to extrahepatic bile duct injury)
- 34. Liver rock: PSC can progress to cirrhosis (due to intrahepatic bile duct injury)
- 35. **Child with dilated biliary gourd**: choledochal cysts are dilations of the bile duct (congenital, present in childhood)
- 36. Hitting abdomen: choledocal cysts can cause abdominal pain
- 37. Yellow kid: choledocal cysts can cause jaundice
- 38. Gourd held under right arm: choledocal cysts can cause a RUQ mass
- 39. Crab in biliary tree : choledochal cysts increase the risk of cholangiocarcinoma
- 40. **Sea-GALL stones**: choledochal cysts increase the risk of gallstones (due to obstruction of bile ducts and cholestasis
- 41. **Crab grasping biliary seaweed**: cholangiocarcinoma is adenocarcinoma of bile duct epithelial cells (can be intra- or extrahepatic)
- 42. **Orca totem pole**: the liver fluke Clonorchis sinensis is a risk factor for cholangiocarcinoma
- 43. **Dark yellow water**: cholangiocarcinoma obstructing the biliary tree can cause light stools and dark urine
- 44. Palpable seaGALL: cholangiocarcinoma can present with Courvoisier sign (non-tender palpable gallbladder and jaundice)
- 45. **Thin and sweaty with flame hadband**: cholangiocarcinoma can present with weight loss, fever, and night sweats
- 46. **Glandy sea foam**: on histology, cholangiocarcinoma presents with glands and mucin

Hepatobiliary 2.1 - Cirrhosis - Pathogenesis & Clinical Manifestations





- 1. Stone liver: cirrhosis (irreversible fibrosis of the liver)
- 2. Alcoholic: chronic alcohol use is the most common cause of cirrhosis
- 3. Hippo armor: chronic viral hepatitis (Hep C and B) can cause cirrhosis
- 4. **Antibody arrows**: autoimmune diseases (e.g. autoimmune hepatitis, primary biliary cirrhosis, primary sclerosing cholangitis) can cause cirrhosis
- 5. AA shears: alpha-1 antitrypsin deficiency (AAT) can cause cirrhosis
- 6. Copper Kayser-Fleischer rings: Wilson disease can cause cirrhosis
- 7. Iron weight: hemochromatosis can cause cirrhosis
- 8. "The Space of Disse": the perisinusoidal space (space of Disse) is located between the sinusoids (composed of endothelial cells, carry blood) and hepatocytes
- 9. **Stars in the space** : stellate cells in the perisinusoidal space activate to become myofibroblasts [in cirrhosis]
- 10. **Fibrotic shrubbery** : myofibroblasts (activated stellate cells) deposit disorganized collagen in perisinusoidal space → fibrosis [in cirrhosis]
- 11. **Blossoming lilies**: liver stem cells create new hepatocytes in response to cell injury [in cirrhosis]
- 12. **Regenerative fountains**: regenerative nodules consist of islands of proliferating hepatocytes [in cirrhosis]
- 13. **Shrubbery around fountains** : collagen deposition in perisinusoidal space \rightarrow bands of fibrosis surround regenerative nodules [in cirrhosis]
- 14. **Compressed sinusoidal snake** : widespread fibrosis → compression of sinusoids → increased intrasinusoidal pressure [in cirrhosis]
- 15. **Cracked PORThole**: increased intrasinusoidal pressure (due to fibrosis) → increased pressure in portal venous system (portal hypertension)
- 16. **Diamond pattern**: normal sinusoids have fenestrations that permit exchange of solutes between blood and hepatocytes (facilitates detoxification of blood)
- 17. **Shrinking diamond pattern**: in a cirrhosis, fibrosis creates a thicker extracellular matrix \rightarrow shrinks fenestrations \rightarrow decreased exchange of solutes between blood and hepatocytes \rightarrow decreased detoxification of blood
- 18. **Blood dripping from stone pot**: cirrhosis can cause coagulopathy (easy bruising, prolonged bleeding after injury, bleeding into joints, hemorrhagic stroke) due to decreased synthesis of clotting factors [impaired synthetic function of liver]

- 19. Raised ParaTrooper: coagulopathy in liver disease causes a prolonged prothrombin time (PT) and elevated INR
- 20. **Zeus extrinsically flying into scene**: PT measures the EXTRINSIC clotting pathway (abnormal in early liver dysfunction due Factor VII (protein with shortest half-life)
- 21. **Falling photo album**: cirrhosis can cause hypoalbuminemia (due to decreased synthesis of albumin) [impaired synthetic function of liver]
- 22. **Poofy pants**: cirrhosis can cause peripheral edema (hypoalbuminemia → decreased serum oncotic pressure) (LATE marker of liver dysfunction)
- 23. **Falling bones**: cirrhosis can cause decreased TOTAL serum calcium levels (hypoalbuminemia → decreased calcium bound to albumin)
- 24. "I" bone not falling: in cirrhosis, IONIZED serum calcium is normal (therefore no symptoms of hypocalcemia, despite decreased TOTAL serum calcium)
- 25. **Broken sun pots** : cirrhosis can cause Vitamin D deficiency (decreased synthesis of 25-hydroxylase → decreased levels of 25-OH Vitamin D)[impaired synthetic function of liver]
- 26. Falling candy: cirrhosis can cause fasting hypoglycemia (due to decreased glycogenolysis and gluconeogenesis)
- 27. **Collard billygoat** : in cirrhosis, impaired secretion of conjugated bilirubin into bile \rightarrow conjugated hyperbilirubinemia [impaired detox function of liver]
- 28. **Un-collared billygoat**: in severe cirrhosis, decreased conjugation of bilirubin in the liver \rightarrow unconjugated hyperbilirubinemia (in addition to conjugated hyperbilirubinemia)
- 29. Yellow goat : in cirrhosis, increased serum bilirubin \rightarrow jaundice
- 30. **NH3 spray bottle**: in cirrhosis, decreased metabolism of ammonia via the urea cycle → increased serum ammonia and decreased serum urea [impaired detox function of liver]
- 31. Clutching head : in cirrhosis, increased serum ammonia crosses blood-brain barrier \rightarrow hepatic encephalopathy
- 32. **Swirly eyes**: hepatic encephalopathy can manifest as altered mental status, seizures, coma, or even death
- 33. **Pegasus's flapping wings**: hepatic encephalopathy can present with asterixis (flapping tremor of the hands)
- 34. **Aromatic benzene armor**: hepatic encephalopathy results from conversion of aromatic amino acids (phenylalanine, tryptophan) into false neurotransmitters (instead of metabolization into urea)

Hepatobiliary 2.1 - Cirrhosis - Pathogenesis & Clinical Manifestations





- 35. **Female symbol**: in cirrhosis, decrease metabolism of estrogen and androstenedione → increased serum estrogen (androstenedione is aromatized into estrogen by adipose tissue) [impaired detox function of liver]
- 36. **Red gloves**: in cirrhosis, elevated serum estrogen causes palmar erythema
- 37. **Two round shields**: in men with cirrhosis, elevated serum estrogen causes gynecomastia
- 38. **Floppy sword** : in men with cirrhosis, elevated serum estrogen \rightarrow increased sex-hormone binding globulin \rightarrow decreased free testosterone \rightarrow erectile dysfunction
- 39. **Red spider**: in cirrhosis, elevated serum estrogen causes vascular telangiectasis ("spider angiomas") on trunk, face, and arms
- 40. **PORThole in the cave**: the portal venous system meets the systemic venous system at sites of portacaval anastomosis (distal esophagus, distal rectum, superficial veins of abdominal wall)
- 41. **Snakes in throat**: portal hypertension can cause esophageal and gastric varices
- 42. **Snakes from gluteal pillar**: portal hypertension can cause hemorrhoids
- 43. **Medusa's head**: portal hypertension can cause engorged paraumbilical veins ("caput medusae")
- 44. **Blood spilling from mouth**: in portal hypertension, esophageal and gastric varices can rupture → massive hematemesis
- 45. Large spleen shield : portal hypertension causes splenomegaly and splenic dysfunction
- 46. **Broken armor plates** : in portal hypertension, splenic dysfunction \rightarrow sequestration of platelets \rightarrow thrombocytopenia
- 47. **Increased midsection pressure**: cirrhosis can cause ascites (increased portal pressure squeezes fluid into space of Disse → overwhelms lymphatic system → fluid leaks into peritoneal cavity
- 48. **Enflamed tail around abdomen**: ascites can cause spontaneous bacterial peritonitis (SBP, protein-rich ascites fluid breeds bacteria) (presents with fever, abdominal pain, altered mental status)
- 49. **Pink rattle tail**: SBP is usually caused by E. coli or other gram negative rods
- 50. Falling helmet: SBP can cause altered mental status

- 51. **Rain umbrella shield**: cirrhosis can lead to activation of the reninangiotensin-aldosterone system (decreased intravascular volume → decreased cardiac output → RAAS activation) (exacerbated by liver unable to metabolize aldosterone)
- 52. **Falling bananas**: cirrhosis can cause hypokalemia (liver unable to metabolize aldosterone → increased renal exchange of sodium for potassium → decreased serum potassium)
- 53. Falling peanuts : cirrhosis can cause hyponatremia (decreased intravascular volume \rightarrow secretion of antidiuretic hormone [ADH] \rightarrow water retention dilutes serum sodium)
- 54. **Cracked kidney canteen**: severe cirrhosis can cause hepatorenal syndrome (renal failure without primary kidney disease)
- 55. **Dilated splanchnic snake**: cirrhosis can cause vasodilation of splanchnic (GI) arteries [in hepatorenal syndrome]
- 56. **Gaseous fog**: vasodilation of splanchnic arteries is due to increased nitric oxide (decreased number of Kupffer macrophages in the liver \rightarrow higher levels of bacterial DNA in blood \rightarrow release of nitric oxide) [in hepatorenal syndrome]
- 57. Rain umbrella : cirrhosis can cause activation of renin-angiotensin-aldosterone system (RAAS)
- 58. **Angiotensin suspenders**: elevated angiotensin II causes systemic vasoconstriction (including renal vessels) → decreased renal blood flow [in hepatorenal syndrome]
- 59. **Empty kidney canteen**: decreased renal-blood flow → pre-renal kidney injury [in hepatorenal syndrome]
- 60. **"End O' The Line"**: in hepatopulmonary syndrome, the liver is unable to metabolize vasodilators (e.g. endothelin)
- 61. **Dilated pulmonary capillary snake**: increased nitric oxide and endothelin → vasodilation of pulmonary capillaries → ventilation-perfusion (V/Q) mismatch [in hepatopulmonary syndrome]

Hepatobiliary 2.2 - Hereditary Hemochromatosis & Wilson's Disease





- Big iron weight: hereditary hemochromatosis is a disorder of iron overload (results in increased iron deposition in organs (liver, pancreas, pituitary, heart, etc.))
- 2. 50 lb weight: hemochromatosis is most commonly diagnosed in men in their 50's
- 3. "Pause": in women, hemochromatosis is usually diagnosed after menopause (because premenopausal women have low iron states from menses, pregnancy, breastfeeding
- 4. Shy kid: hereditary hemochromatosis is an autosomal recessive condition
- 5. **Six-pack**: hereditary hemochromatosis is most commonly caused by a missense mutation in the HFE gene on chromosome 6
- 6. Venice BeacH, LA, American Athletic Association : the HFE gene is linked to the HLA-A3 locus
- 7. Iron hitting ${f gut}$: in hemochromatosis, enterocytes expressing HFE mutation cause upregulated absorption of iron from the GI tract
- 8. Overwhelming transferrin bar: in hemochromatosis, loading of iron onto transferrin by enterocytes is upregulated
- 9. Distracted trainer : in hemochromatosis, liver does not sense iron overload \rightarrow low hepcidin
- 10. Fallen iron keys: hepcidin (usually responsible for locking iron inside enterocytes and out of the circulation) is LOW in hemochromatosis
- 11. **Fe deposited on liver**: in hemochromatosis, iron overload leads to iron deposits in liver and other organs (pancreas, skin, heart, pituitary, joints, etc.)
- 12. **Golden sand granules**: in hemochromatosis, iron deposits in organs as hemosiderin (appears as golden yellow granules in cytoplasm of cells)
- 13. **Oxidizing sparks**: in hemochromatosis, excess iron creates hydroxyl free radicals → cellular damage
- 14. Fibrous liver tree : hemochromatosis can cause liver fibrosis (cellular damage repair process \rightarrow collagen deposition and scarring
- 15. Liver stone: hemochromatosis can cause cirrhosis
- 16. Cancer crabs: hemochromatosis increases the risk of hepatocellular carcinoma (via progression to cirrhosis)
- 17. **Bronzed skin**: hereditary hemochromatosis can cause bronze hyperpigmentation (iron deposits in skin → melanin production)
- 18. Elevated candy dumbbell : hereditary hemochromatosis can cause diabetes mellitus (iron produces free radicals → destruction of beta islet cells)("bronze diabetes")
- Muddy puddle with debris : hereditary hemochromatosis can cause steatorrhea and malabsorption (free radicals injure exocrine pancreas → insufficient pancreatic digestive enzymes)
- 20. **Dilated heart punching bag**: hemochromatosis can cause DILATED cardiomyopathy (due to iron deposition in myocardium)
- 21. Heart tattoo under mesh top: hemochromatosis can cause RESTRICTIVE cardiomyopathy (due to iron deposition in myocardium)
- 22. Pituitary punching bag : hemochromatosis can cause iron deposition in anterior pituitary \rightarrow decreased sex hormone production
- 23. **Tiny male speedo**: hemochromatosis can cause secondary hypogonadism (due to iron deposition in anterior pituitary)
- 24. **Joint wraps** : hemochromatosis can cause degenerative joint disease (iron deposition in joints \rightarrow chondrocalcinosis)

- 25. Barbell up high: in hemochromatosis, serum iron and ferritin levels are $\ensuremath{\mathsf{INCREASED}}$
- 26. **Broken transferrin bars** : in hemochromatosis, total iron binding capacity (TIBC) is LOW (increased binding of iron to transferrin → decreased synthesis of transferrin → decreased TIBC)
- 27. Saturated transferrin barbell : in hemochromatosis, transferrin saturation is elevated (best initial test for screening)
- 28. **Blue face paint**: in hemochromatosis, liver biopsy shows golden granules of hemosiderin in the cytoplasm (stain BLUE with prussian blue stain)
- 29. Bleeding pipe: phlebotomy is a treatment for hemochromatosis
- 30. **Grabby sandbox toy**: deferoxamine is a treatment for hemochromatosis (chelates iron)
- 31. **Transfusing blood**: frequent blood transfusions (e.g. sickle cells anemia, thalassemia major) can cause SECONDARY hemochromatosis (hemosiderosis)
- 32. **Poorly assembled hemoglobin weights**: ineffective erythropoiesis (e.g. betathalassemia) can cause iron overload (hemosiderosis)
- 33. **Pile of copper pennies**: Wilson disease (hepatolenticular degeneration) is a disease of copper overload)
- 34. Shy kid: Wilson disease is autosomal recessive
- 35. "7B" vending machine with 3 P batteries: Wilson disease is due a mutation in hepatocyte copper-transporting ATPase (ATP7B)
- 36. **Biliary tree**: in Wilson disease, hepatocytes are unable to transport copper into bile for excretion
- 37. **Dropped cerulean blue shorts** : in Wilson disease, serum ceruloplasmin (normally transports copper in blood) is LOW \rightarrow increased free serum copper \rightarrow copper deposits in tissues
- 38. Copper ring: Wilson disease can cause Kayser-Fleischer rings (copper-colored rings encircling periphery of iris)
- 39. **Oxidizing sparks**: in Wilson disease, copper accumulates in hepatocytes and causes formation of free radicals → cellular damage
- 40. Large liver: Wilson disease can cause hepatosplenomegaly
- 41. Stony liver: Wilson disease can cause liver fibrosis → cirrhosis
- 42. **Broken red buckets**: Wilson disease can cause hemolytic anemia (due to excess circulating copper)
- 43. **Protruding tongue**: Wilson disease can cause dysarthria (due to copper deposits in the brain)
- 44. ${\bf Cog\ wheel}$: Wilson disease can cause Parkinsonian symptoms (i.e. cogwheel rigidity)
- 45. **Shaking arm**: Wilson disease can cause movement disorders (e.g. dystonia, tremors, choreoathetosis)
- 46. Confusion: Wilson disease can cause dementia
- 47. **Dropped flyer "SERIOUS about fitness at TOTAL gym"**: in Wilson disease, TOTAL SERUM copper is LOW (due to LOW ceruloplasmin levels)
- 48. Free floating pennies: in Wilson disease, FREE serum copper is ELEVATED
- 49. Yellow penny puddle: in Wilson disease, free URINE copper is ELEVATED
- 50. **Grabby pencil crane**: Wilson disease is treated with penicillamine (copper chelator that inhibits absorption in the intestines)

Hepatobiliary 2.3 - Alcoholic and Non-alcoholic Fatty Liver Disease





- 1. Acetaldehyde stool after alcohol: ethanol is metabolized to acetaldehyde in the liver
- 2. **Wiping-up ethanol** : alcohol dehydrogenase converts ethanol to acetaldehyde in the liver
- 3. NAD+ electrolyte drink: conversion of ethanol to acetaldehyde involves oxidation, which requires NAD+ (reduced to NADH)
- 4. Cracked beta fish-tank: excessive alcohol intake results in depletion of NAD+ → insufficient NAD+ for fatty acid beta oxidation → impaired oxidation of fatty acids in the liver
- 5. **Overweight patrons**: in heavy alcohol use, impaired oxidation of fatty acids leads to buildup of fatty acids in the liver
- 6. **Hung over on seat**: a "hangover" (headache, nausea, etc) is caused by the buildup of acetaldehyde
- 7. Acetate stool under NAD+ : acetaldehyde dehydrogenase converts acetaldehyde to acetate (via oxidation, requires NAD+ \rightarrow NADH)
- 8. Chubby lounge singer on liver piano : repeated ingestion of ethanol results in a chronic shortage of NAD+ → fatty acids build up in the liver → fatty change (steatosis)
- 9. **Polka dots**: on histology, steatosis causes hepatocytes to fill with vesicles of fat (MICROvesicular steatosis if small globules of fat, MACROvesicular steatosis if large globules of fat displacing nucleus)
- 10. "Ill" course dessert in center: zone 3 (the centrilobular zone) is the first area to develop fatty change (also the most sensitive to ischemia and metabolic toxins
- 11. Sending back order : fatty liver (steatosis) is reversible (cessation of alcohol)
- 12. Flaming liver piano : persistent steatosis can lead to steatohepatitis (reactive oxygen species, direct toxic effect of ethanol/acetaldehyde)
- 13. CytoCOINS : hepatocyte damage \to cytokine release \to inflammatory infiltrate (alcoholic hepatitis)
- 14. First responders: an influx of neutrophils occurs in alcoholic hepatitis
- 15. **Filamentous lace** : alcoholic hepatitis damages intermediate filaments→ filament clumping
- 16. **Eosinophilic splotches of wax**: clumped intermediate filaments appear as pink densities ("Mallory bodies") within hepatocytes (in alcoholic hepatitis)
- 17. **Balloons**: damage to hepatocytes can cause them to balloon (in alcoholic hepatitis)
- 18. **Popped balloon**: damaged ballooned hepatocytes can progress to hepatocyte necrosis (in alcoholic hepatitis)
- 19. **Fibrous plant around central column**: fibrosis starts near the central vein of the liver (in zone 3, the same location as the start of steatosis) (occurs in alcoholic hepatitis)
- 20. **Midsection and hair on fire**: alcoholic hepatitis can present with a large painful liver and fever/leukocytosis
- 21. "toAST" 2 glasses : in alcoholic hepatitis, hepatocytes leak enzymes \rightarrow elevated AST and ALT (AST > ALT, 2:1 ratio)
- 22. **Gadget knife**: gamma glutamyl transpeptidase (GGT) is elevated in chronic alcohol use (including alcoholic hepatitis)

- 23. **Dousing liver flame** : alcoholic hepatitis is generally reversible
- 24. Scarred liver coral: alcoholic hepatitis may progress to cirrhosis (scarring that forms septa and nodules, generally irreversible)
- 25. Cancer crab: cirrhosis can cause hepatocellular carcinoma
- 26. **Sober lounge singer**: non-alcoholic fatty liver disease (NAFLD) is similar to alcoholic fatty liver disease, and can progress to steatohepatitis and cirrhosis
- 27. **Polka dots**: on histology, steatosis causes hepatocytes to fill with vesicles of fat (MICROvesicular steatosis if small amount of fat, MACROvesicular steatosis if large amount of fat displacing nucleus)
- 28. Extra-chubby singer : excessive visceral adipose tissue is likely the cause of NASH (visceral adipose \rightarrow insulin resistance \rightarrow altered fatty acid metabolism \rightarrow liver retains more fatty acids)
- 29. Flaming liver with first responders: nonalcoholic steatohepatitis (NASH) is similar to alcoholic steatohepatitis (both inflammatory conditions with neutrophil infiltration)
- 30. **Hepatocyte ballooning**: NASH can cause hepatocytes to balloon (similar to alcoholic hepatitis)
- 31. **Eosinophilic splotches of wax**: NASH can cause pink densities ("Mallory bodies") within hepatocytes (similar to alcoholic hepatitis)
- 32. **Metabolic brownies**: metabolic syndrome (at least 2 of obesity, insulin resistance, dyslipidemia, hypertension) is a risk factor for progression of NAFLD to NASH to liver fibrosis
- 33. "mALT" shake : In NASH, both ALT and AST are elevated (usually ALT greater than AST)
- 34. **Scarred liver coral, again**: non-alcoholic steatohepatitis may progress to cirrhosis (scarring that forms septa and nodules, generally irreversible)(similar to alcoholic hepatitis)
- 35. Rays of sun: Reye syndrome (triad of encephalopathy, liver microvesicular fatty change, and elevated serum transaminases) in children who take aspirin
- 36. **Aspirin umpire and viral lanterns**: Reye syndrome is associated with aspirin administration to young children with a viral infection (e.g chicken pox, upper respiratory infection)
- 37. Blowing nose : Reye's syndrome may be preceded by symptoms of view illness
- 38. Cracked mitochondrial window : Reye syndrome is a result of mitochondrial damage \rightarrow impaired β -oxidation of fatty acids \rightarrow accumulation of fatty acids, inflammation, elevated transaminases
- 39. Little droplets on liver mat: Reye syndrome causes MICROvesicular fatty changes (small globules of fat in hepatocyte cytoplasm)
- 40. "mALT" shake : in Reye syndrome, hepatocyte damage leads to elevated ALT and AST
- 41. **NH3 spray** : in Reye syndrome, mitochondrial injury \rightarrow impaired urea cycle \rightarrow decreased metabolism of ammonia \rightarrow elevated serum ammonia levels
- 42. **Hitting kid in the brain hat**: Reye syndrome causes encephalopathy (initially somnolence, regression of motor development) due to elevated serum ammonia levels
- 43. **Wet head**: in Reye syndrome, cerebral edema and worsening encephalopathy may develop (seizures, vomiting, upper motor neuron signs)

Hepatobiliary 2.4 - Liver Tumors & Hepatocellular Carcinoma





- 1. Focal tesla node: Focal Nodular Hyperplasia (FNH) is the most common benign, non-vascular liver mass
- 2. **Solitary star shape**: FNH is a solitary area of hepatocyte proliferation (forms a nodular star pattern)
- 3. Red wire around node : FNH has a large central artery
- 4. **Spoked wires**: in FNH, contrast imaging shows a "spoke wheel" vascular pattern
- 5. **Green wire around node**: FNH contains bile ductules, arterioles, and portal venules
- 6. Female assistant: FNH, hepatic hemangiomas, and hepatic adenomas are most common in reproductive age women
- 7. Estrogen earrings: estrogen stimulates growth of FNH and hepatic adenomas
- 8. **Pregnant woman**: pregnancy stimulates growth of FNH and hepatic adenomas (due to increased estrogen levels)
- 9. **OCP platter**: OCPs stimulate growth of FNH and hepatic adenomas (due to increased estrogen levels)
- 10. 10Cavern containing red cord : hepatic hemangioma (vascular liver tumor containing "cavernous" dilated cystic areas)
- 11. #1 finger: hepatic hemangiomas are the most common benign liver mass
- 12. **Dilated fragile red wires**: in hepatic hemangiomas, dilated vascular spaces filled with blood are lined by a single layer of epithelium (thin adventitia and no smooth muscle)
- 13. Clumped red dots within dilated wires: on histology, hepatic hemangiomas show dilated cystic spaces filled with red blood cells
- 14. Bloody wires: hepatic hemangiomas are prone to hemorrhage
- 15. **Pain lines**: hepatic hemangiomas can cause acute RUQ pain (due to hemorrhage)
- 16. **Bloody grabber**: fine needle aspiration or biopsy of hepatic hemangiomas will result in profuse bleeding
- 17. ADDing to liver: hepatic adenoma (benign liver mass)
- 18. **Male symbol needle** : anabolic androgens stimulate the growth of hepatic adenomas
- 19. "Igierke" : type 1 (Von Gierke's disease) and type 3 glycogen storage diseases can cause hepatic adenomas
- 20. Branched pink hair: on histology, hepatic adenomas are composed of large hepatocytes full of glycogen
- 21. Red sutures: hepatic adenomas contain thin-walled vessels and sinusoids WITHOUT bile ducts
- 22. Massive back hump: hepatic adenomas can be large (up to 30 cm)
- 23. Bloody cylinder : hepatic adenomas can rupture \rightarrow massive hemorrhage
- 24. Pain lines : hepatic adenomas can cause acute RUQ pain (due to hemorrhage)
- 25. **Lightning sign**: hepatic adenomas can cause hypovolemic shock (due to massive hemorrhage)
- 26. Cancer crab : hepatic adenomas carry a small risk of transformation to HCC
- 27. #1 Pro-Metastasis": the majority of malignancies in the liver are metastases
- 28. Transplanted lung and colon tissue : liver metastases most commonly originate from the colon or lungs $\,$
- 29. Cancer crab claws : hepatocellular carcinoma (HCC) is the most common primary liver cancer

- 30. Stone liver: cirrhosis is the most common cause of HCC
- 31. **Pouring "XXX" bottle** : alcoholic liver disease (and non-alcoholic fatty liver disease) can cause cirrhosis → may lead to HCC
- 32. Large iron weight: iron overload in hereditary hemochromatosis can cause HCC (via progression to cirrhosis)
- 33. **Antibody bolts**: autoimmune liver diseases (autoimmune hepatitis, primary biliary cholangitis, primary sclerosing cholangitis) can cause HCC (via progression to cirrhosis)
- 34. "AA Trimming" shears : α -1 antitrypsin deficiency can cause HCC (via progression to cirrhosis)
- 35. Oversized hippo: hepatitis B is the most common cause of HCC worldwide
- 36. **Hippo under stone liver** : viral hepatitis (hepatitis B, hepatitis C) can lead to HCC (usually via progression to cirrhosis)
- 37. **Peace sign & DNA double helix**: hepatitis B infection CAN cause HCC WITHOUT cirrhosis (due to integration into hepatocyte DNA) but USUALLY occurs via progression to cirrhosis
- 38. "Toxin" on scarecrow: exposure to AFLATOXIN produced by Aspergillus can cause HCC (WITHOUT cirrhosis, by causing a mutation in p53 tumor suppressor gene)
- 39. **Scarecrow leaking wheat**: Aspergillus grows on dry food products (grain, corn, soybeans, peanuts)
- 40. **AZO dye beakers**: chronic exposure to azo dyes (paints, leather working) increases the risk of HCC
- 41. **Green canaliculi catheters** : well-differentiated ("organized") HCC tumors have bile-containing pseudo-canaliculi on histology
- 42. Oversized eyeballs with large purple centers: poorly differentiated "disorganized" HCC contain "giant cells" (large hepatocytes with multiple enlarged nuclei)
- 43. **Crumbling liver**: HCC commonly presents with acute liver decompensation (encephalopathy, worsening or bloody ascites, increased jaundice, fever, spontaneous bacterial peritonitis)
- 44. **Cork obstructing neck of pressurized blue liver**: HCC can occlude hepatic veins → leading to venous congestion and portal hypertension (Budd-Chiari syndrome) (presents with abdominal pain, ascites, and enlarged liver)
- 45. Liver juice splashing into lung cylinder: HCC most commonly metastasizes to the lungs
- 46. $\mbox{\bf ALF's}$ Fresh Produce bag : serum alpha-fetoprotein (AFP) is a tumor marker for HCC
- 47. Tangled red hair & crab claw : angiosarcoma (malignant neoplasm of hepatic vascular endothelial cells)
- 48. "CD" belt & "XXXI" stitching : angiosarcomas express CD31
- 49. Bloody dress : angiosarcomas are prone to rupture \rightarrow massive hemorrhage
- 50. **ARSENIC**: exposure to arsenic (pesticides, herbicides, wood, mining, glass making, etc) is a risk factor for hepatic angiosarcoma
- 51. **PVC pipe**: exposure to vaporized polyvinyl chloride (PVC) (a component of plastic pipes) is a risk factor for liver angiosarcoma
- 52. **Glowing "Th"**: exposure to Thorotrast (radioactive thorium dioxide, an early radiocontrast dye) is a risk factor for hepatic angiosarcoma

Hepatobiliary 3.1 - Acute & Chronic Pancreatitis



- 1. Pancreatic sea sponge: pancreatitis can be acute or chronic
- 2. **Protease meat cleaver**: trypsin is a protease that digests dietary proteins and cleaves and activates zymogens (e.g. trypsinogen)
- 3. **AUTOmatic meat knife** : injury to the pancreas or obstruction of the pancreatic ducts → premature activation of trypsinogen → activation of other pancreatic enzymes → acute pancreatitis
- 4. **Roasting lipid-laden pig** : release of the pancreatic enzyme lipase \rightarrow autodigestion of adipose tissue \rightarrow fat necrosis (visible on histology in pancreatitis)
- 5. Fat dripping on calcified bones : fatty acids bind to calcium ions → calcium precipitations (visible on histology in pancreatitis)
- 6. **Sea-gall stones**: gallstones are the most common cause of acute pancreatitis (due to common bile duct obstruction)]
- 7. Stone on common seaweed stem : gallstone obstruction of the common bile duct (choledocolithiasis) \rightarrow acute pancreatitis
- 8. **Alcoholic**: chronic alcohol abuse is the second most common cause of acute pancreatitis (due to direct toxic injury and increased viscosity of pancreatic secretions)
- 9. Raised trident fork: hypertriglyceridemia can cause acute pancreatitis (more common at levels >1000mg/dL)
- 10. **Family of raised tridents** : familial hyperlipidemia syndromes can cause extreme hypertriglyceridemia and pancreatitis (e.g. type I high chylomicrons; type IV high VLDL; type V high chylomicrons and VLDL)
- 11. Mortar and pestle: medications can cause acute pancreatitis
- 12. "Seatbelt" arm sling: trauma (especially seatbelt trauma) can cause acute pancreatitis
- 13. **Periscope inspecting sponge**: endoscopic retrograde cholangiopancreatography (ERCP) can cause acute pancreatitis
- 14. Sharp limbo stick; acute pancreatitis presents with sharp epigastric pain :
- 15. Back pain: epigastric pain radiates to the back (in pancreatitis)
- 16. Nauseated: acute pancreatitis presents with nausea and vomiting
- 17. Flame bandana: acute pancreatitis presents with fever
- 18. **Breaking down sugary apple**: serum amylase is elevated in acute pancreatitis (not specific because amylase is also produced in the salivary glands)
- 19. Fatty juice : serum lipase is elevated in acute pancreatitis (more specific than amylase)
- 20. **Dropping CALCI-YUM ice cream**: severe pancreatitis may present with hypocalcemia (due to free fatty acids binding up calcium)
- 21. **LOW TIDE**: in severe acute pancreatitis inflammation causes accumulation of fluid around the pancreas (third-spacing) \rightarrow decreased intravascular volume (hypovolemia)

- 22. "Sea sALT rub for rib roASTs": in gallstone pancreatitis, obstruction of the common bile duct → liver inflammation → elevated ALT and AST (particularly ALT)
- 23. To ASTing with alcohol : in alcohol-induced pancreatitis, AST: ALT ratio may be > 2:1
- 24. Big red flowers: alcohol abuse may cause macrocytosis (MCV >100)
- 25. **Fibrous coconut shell**: acute pancreatitis may present with a pseudocyst (liquified pancreatic tissue due to autodigestion is walled off by a capsule of fibrous granulation tissue \rightarrow NOT a true cyst)
- 26. **Death tiki**: severe acute pancreatitis may progress to acute necrotizing pancreatitis (decreased blood flow to the acinar cells → further autodigestion)
- 27. White chalky tiki skulls: in acute necrotizing pancreatitis, gross pathology shows white chalky areas of fat necrosis (in pancreas, surrounding retroperitoneal fat, omentum, or mesentery)
- 28. **Tiki blood**: acute necrotizing pancreatitis can cause hemorrhage (appears as red-black areas on gross pathology)
- 29. **ARDS shark**: pancreatitis can cause acute respiratory distress syndrome (ARDS) (pancreatic autodigestion → release of phospholipase → breakdown of surfactant in the lungs)
- 30. **Splenic jellyfish obstructed by fibrous hula skirt**: pancreatitis can cause splenic vein thrombosis
- 31. **Alcoholic**: chronic alcohol abuse is the most common cause of chronic pancreatitis
- 32. "IDIOT": many cases of chronic pancreatitis are idiopathic
- 33. Thick CF sap: cystic fibrosis can cause chronic pancreatitis
- ${\bf 34. \ Shriveled \ pancreas \ sponge: long-term \ chronic \ pancreatitis \ leads \ to \ fibrosis, \ calcification, \ and \ atrophy}$
- 35. Calcified white flowers: chronic pancreatitis can present with calcified concretions throughout the pancreas (visible on CT and ultrasound)
- 36. **Fibrous coconut shell**: chronic pancreatitis can present with pseudocysts (also present in acute pancreatitis)
- 37. Frothy puddle : chronic pancreatitis can cause steatorrhea (loss of pancreatic acini \rightarrow decreased lipase secretion by the pancreas \rightarrow increased lipids in GI tract
- 38. Falling A-DECK : chronic pancreatitis can cause deficiencies in the fat soluble vitamins ADE and K
- 39. Candy lanterns: chronic pancreatitis can cause destruction of islets of langerhans \rightarrow loss of endocrine function, including decreased insulin secretion \rightarrow diabetes \rightarrow elevated blood glucose
- 40. Cancer crabs: chronic pancreatitis increases the risk of pancreatic adenocarcinoma

Hepatobiliary 3.2 - Pancreatic Cancer & Islet Cell Tumors



- Cancer crabs: pancreatic cancer can arise from pancreatic glands (adenocarcinoma) or from endocrine cells (neuroendocrine tumors)
- 2. **Male smoker**: smoking is a risk factor for pancreatic adenocarcinoma (also more common in males)
- 3. **Shriveled pancreas sponge**: chronic pancreatitis is a risk factor for pancreatic adenocarcinoma
- 4. **Alcoholic**: chronic alcohol abuse is the most common cause of chronic pancreatitis
- 5. Calcified white flowers: chronic pancreatitis often presents with concretions of calcified fat necrosis)
- 6. **Broccoli**: germline mutations in the tumor suppressor genes BRCA1 and 2 are the most common cause of familial pancreatic adenocarcinoma
- 7. **Family photo**: patients with BRCA 1 and 2 mutations may have a family history of cancer (e.g. breast, ovarian)
- 8. Fight cancer ribbon: BRCA1 and 2 are tumor suppressor genes
- 9. Cancerous rats: mutations in the KRAS proto-oncogene are a risk factor for pancreatic adenocarcinoma (present in almost all pancreatic adenocarcinomas)
- 10. **CoDe book N2A**: mutations in the CDKN2A tumor suppressor gene are a risk factor for pancreatic adenocarcinoma
- 11. $\bf HMS~SalaMAnDer$: mutations in the SMAD4 tumor suppressor gene are a risk factor for pancreatic adenocarcinoma
- 12. **Fight cancer ribbon**: CDKN2A and SMAD4 and are tumor suppressor genes
- 13. Fish heads : the majority of pancreatic adenocarcinomas arise from the head of the pancreas ${\bf r}$
- 14. Stepping on common seaweed trunk : masses at the head of the pancreas obstruct the common bile duct \rightarrow jaundice (in pancreatic adenocarcinoma)
- 15. **Yellow jacket**: masses at the head of the pancreas can cause jaundice (in pancreatic adenocarcinoma)
- 16. **Light bird poop**: obstruction of common bile duct (by mass at head of pancreas) → decreased (conjugated) bilirubin in GI tract → light or clay colored stools (in pancreatic adenocarcinoma)
- 17. **Dark water**: obstruction of common bile duct (by mass at head of pancreas) → elevated conjugated bilirubin in blood → conjugated bilirubin filters into kidneys → dark urine (in pancreatic adenocarcinoma)

- 18. **Painless sea-gall**: mass at head of pancreas can cause a palpable, nontender gallbladder (usually associated with jaundice and called Courvoisier's sign) (in pancreatic adenocarcinoma)
- 19. **Yellow jacket**: painless jaundice is a relatively early sign in tumors arising from the pancreatic head (in pancreatic adenocarcinoma)
- 20. **Hit by crab cages in front and back**: pancreatic adenocarcinoma commonly presents with epigastric pain radiating to the back
- 21. **Thin and sweaty**: pancreatic adenocarcinoma can present with constitutional symptoms (weight loss, fever, night sweats)
- 22. **LEFT SUPRACLAVICULAR pigeon**: pancreatic adenocarcinoma may metastasize to the LEFT SUPRACLAVICULAR node ("virchow's node")
- 23. **PERIUMBILICAL pigeon**: a pancreatic adenocarcinoma metastasis can form a PERIUMBILICAL mass (sister mary joseph nodule)
- 24. **Migrating net with thrombotic seaweed**: pancreatic adenocarcinoma can cause superficial migratory thrombophlebitis (Trousseau's sign) (a paraneoplastic syndrome)
- 25. **Cushing's cushion**: pancreatic adenocarcinoma can cause Cushing syndrome (a paraneoplastic syndrome)
- 26. "Lost at CEA CAII 19-9": CA 19-9 and CEA are tumor markers for pancreatic adenocarcinoma
- 27. $\mbox{\rm ISLAND}$: pancreatic NEUROENDOCRINE tumors derive from cells in the ISLETS of Langerhans
- 28. "Welcome INside": INSULINOMAS are the most common pancreatic NEUROENDOCRINE tumor
- 29. " β " binoculars : INSULINOMAS derive from BETA cells in the Islets of Langerhans)
- 30. Falling candy: INSULINOMAS can cause HYPOglycemia (due to excess insulin secretion)
- 31. **Neurological candy injury**: hypoglycemia causes tremors, diaphoresis, and confusion (in INSULINOMAS)
- 32. **Satisfied after eating**: patients with hypoglycemia due to INSULINOMAS feel better after a meal
- 33. **C-shaped fruit rinds**: ENDOgenous insulin (including insulinoma) is associated with increased C-peptide levels (cleaved from proinsulin) but this is NOT true for EXOgenous insulin

Hepatobiliary 3.2 - Pancreatic Cancer & Islet Cell Tumors



- 34. Excess glucagon sweetener: GLUCAGONOMAS cause HYPERGLYCEMIA (due to glucagon stimulating gluconeogenesis)
- 35. "Glucαgon" crate : GLUCAGONOMAS are NEUROENDOCRINE tumors derived from the ALPHA cells of the islets of Langerhans
- 36. **Leaking brown puddle**: GLUCAGONOMAS can cause GI side effects (e.g. diarrhea)
- 37. **Emaciated**: GLUCAGONOMAS can cause weight loss (due to decreased storage of glycogen and fat)
- 38. **Disturbed in glucagon box**: GLUCAGONOMAS can cause neuropsychiatric symptoms (depression, dementia, psychosis, agitation)
- 39. **Thrombotic sticks**: GLUCAGONOMAS can cause venous thromboembolism (e.g DVT or PE)
- 40. **Severe splotchy sunburn**: GLUCAGONOMAS can cause necrolytic migratory erythema (epidermal necrosis causing migrating erythematous plaques, usually affects extremities and perioral areas)
- 41. **Oversized "GAS" tank**: GASTRINOMAS are a NEUROENDOCRINE tumor that lead to hypersecretion of gastrin
- 42. **Duodenal "C" shape curving around pancreatic raft**: GASTRINOMAS may originate from the duodenum or pancreas
- 43. **Leaking acid**: GASTRINOMAS cause excessive secretion of stomach acid (also called Zollinger-Ellison Syndrome)
- 44. **Gurgling acid**: GASTRINOMAS can cause GERD (due to excessive secretion of acid in the stomach
- 45. **Holes in duodenal raft**: GASTRINOMAS cause peptic ulcer disease (usually in the duodenum)
- 46. "J" shaped intestinal raft : GASTRINOMAS may cause ulcers in the jejunum
- 47. **Ineffective patches over holes**: peptic ulcer disease caused by GASTRINOMAS may be unresponsive to conventional therapies
- 48. Brown puddle with food debris : GASTRINOMAS can cause malabsorption (due to overly acidic pH in the GI tract) \rightarrow diarrhea
- 49. **Thickened leaves**: in GASTRINOMAS, upper GI endoscopy shows prominent thickening of gastric folds
- 50. "**Top Secret**": a secretin stimulation test may be performed to diagnose GASTRINOMA (unnecessary if gastrin levels are extremely elevated > 1000)(diagnosis is confirmed if gastrin levels rise after administration of secretin)

- 51. "VIP only": VIPOMAS are NEUROENDOCRINE tumors (VIP = vasoactive intestinal peptide; stimulates secretion of water, electrolytes, and bicarbonate by the small intestine and pancreas)
- 52. **Dripping watery puddle**: VIPOMAS cause large volume watery diarrhea (usually > 1 L per day)("pancreatic cholera")
- 53. Depleted potassium banana peels : in VIPOMAS, large volume diarrhea ightarrow dehydration and hypokalemia
- 54. **Nearly empty gastric toilet**: VIP inhibits gastric acid secretion → achlorhydria (low HCL in the stomach) [in VIPOMAS]
- 55. Leader wearing "P" hat, bow tie, abdominal tattoo : MEN1 (Multiple Endocrine Neoplasia 1) causes pituitary, parathyroid, and pancreatic tumors
- 56. "**\$TOP**": SOMATOSTATINOMAS are NEUROENDOCRINE tumors derived from DELTA cells in the Islets of Langerhans (somatostatin inhibits secretion of multiple hormones [insulin, glucagon, cholecystokinin, gastrin, VIP])
- 57. Brown sea foam puddle with food debris: SOMATOSTATINOMAS inhibit cholecystokinin \rightarrow malabsorption diarrhea
- 58. Sea-GALL stones : SOMATOSTATINOMAS inhibit cholecystokinin \rightarrow increased risk of gallstones
- 59. **Nearly empty gastric tank** : SOMATOSTATINOMAS inhibit gastric acid secretion → achlorhydria (low HCL in the stomach)
- 60. **Candy** : SOMATOSTATINOMAS inhibit insulin more than glucagon → HYPERglycemia
- 61. Octogonal sign: Octreotide (somatostatin analog) inhibits insulin, glucagon, gastrin, and VIP (and therefore SOMATOSTATIN relieves the symptoms of insulinomas, glucagonomas, gastrinomas, and VIPomas

Endocrine 1.1 - Hypothalamic & Pituitary Dysfunction - Etiologies

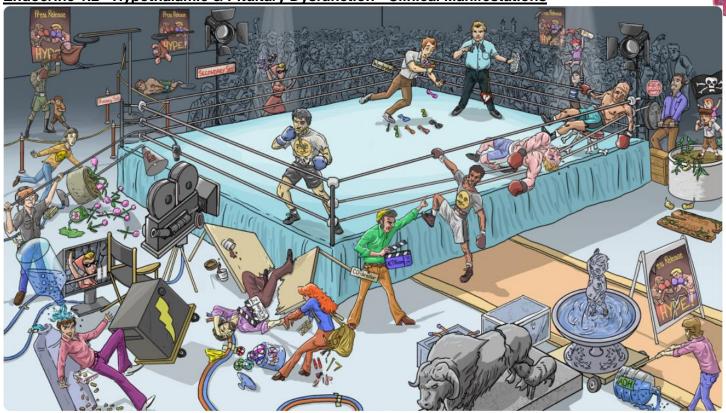




- 1. Punching pituitary bag: hypopituitarism
- 2. "HYPE" Press RELEASE: the HYPOTHALAMUS secretes "releasing hormones" (thyrotropin-releasing hormone [TRH], corticotropin-releasing hormone [CRH])("GO" signals for pituitary)
- 3. Front of punching bag: anterior pituitary
- 4. VASCULAR rope: hormones travel from the HYPOTHALAMUS to the ANTERIOR pituitary through a small VASCULAR network ("hypophyseal portal system") → ANTERIOR pituitary then releases its OWN hormones into the peripheral circulation
- 5. Connecting wires to back of bag: molecules produced by the HYPOTHALAMUS travel via direct neural connection to the POSTERIOR pituitary (NEUROhypophysis) → POSTERIOR pituitary then secretes these molecules into peripheral circulation
- 6. Scuffed-up FLAT PiG: ANTERIOR pituitary dysfunction (HYPOpituitarism) (leads to decreased follicle stimulating hormone [FSH], luteinizing hormone [LH], adrenocorticotropic hormone [ACTH], thyroid stimulating hormones [TSH], prolactin, growth hormone [GH])
- 7. **Torn "HYPE" Press RELEASE** : injury to the HYPOTHALAMUS (traumatic brain injury, brain tumors, radiation therapy, stroke) can decrease secretion of RELEASING hormones from the hypothalamus → HYPOpituitarism
- 8. Cancer crab : brain tumors affecting the HYPOTHALAMUS can cause HYPOpituitarism
- 9. Radiation light : radiation therapy to the HYPOTHALAMUS can cause HYPOpituitarism
- 10. **Stroke of black** : stroke affecting the HYPOTHALAMUS can cause HYPOpituitarism
- 11. Punching in the plus sign : a PITUITARY "ADD" ENOMA (space-occupying lesion in sella turcica → compresses normal pituitary tissue → pressure necrosis → HYPOpituitarism) (most common cause of HYPOpituitarism in adults)
- 12. **Skull gloves**: in a PITUITARY ADENOMA, pressure necrosis causes HYPOpituitarism
- 13. Painful head punch: PITUITARY ADENOMAS can causes headache (due to compression of surrounding structures)
- 14. "X" gloves: PITUITARY ADENOMAS can compress the optic chiasm (location of optic nerve crossing)
- 15. Headgear blocking peripheral vision: PITUITARY ADENOMAS can cause bitemporal hemianopsia (loss of both lateral visual fields) due to compression of optic chiasm
- 16. **Bleeding head**: PITUITARY APOPLEXY (acute spontaneous hemorrhage of the pituitary) (occurs as a result of an enlarging pituitary adenoma)
- 17. Pain lines: PITUITARY APOPLEXY presents with sudden onset of severe headache
- 18. Losing glasses & swirly eyes: PITUITARY APOPLEXY presents with visual field loss and diplopia (double vision)
- 19. **Beat-up punching bag**: PITUITARY APOPLEXY can cause acute HYPOpituitarism
- 20. Losing heart patch: PITUITARY APOPLEXY can cause cardiovascular collapse (due to acute HYPOpituitarism causing decreased ACTH)
- 21. Kid with "CRANIO" headgear: CRANIOPHARYNGIOMA (most common cause of HYPOpituitarism in children)

- 22. "WRATH" with open mouth logo: CRANIOPHARYNGIOMAS are BENIGN neoplasms of the epithelial remnants of RATHKE's pouch (a division of early ORAL ectoderm)
- 23. Scrunchie on ponytail STALK: CRANIOPHARYNGIOMAS most commonly occur in the pituitary STALK (located SUPRASELLAR)
- 24. Pain lines: CRANIOPHARYNGIOMAS can cause headaches (due to mass effect)
- 25. "X" gloves : CRANIOPHARYNGIOMAS can compress the optic chiasm (location of optic nerve crossing)
- 26. **Headgear blocking peripheral vision**: CRANIOPHARYNGIOMAS can cause bitemporal hemianopsia (loss of both lateral visual fields) (due to compression of optic chiasm)
- 27. **Skull gloves**: in CRANIOPHARYNGIOMA, pressure necrosis can cause HYPOpituitarism
- 28. Calcified teeth, and cystic bubbles: CRANIOPHARYNGIOMAS display 3 layers: a calcified component, solid component, and cystic component
- 29. **Leaking turbid fluid**: the cysts of CRANIOPHARYNGIOMAS contain a brown-yellow cholesterol-rich fluid ("machinery oil")
- 30. **Pregnant fighter holding skull bag**: ISCHEMIC NECROSIS of pituitary gland (most commonly due to Sheehan syndrome in postpartum period) causes HYPOpituitarism
- 31. Baby bottles in overloaded estrogen fanny pack : during pregnancy, increased ESTROGEN → hyperplasia of prolactin-secreting cells → increased PROLACTIN → INCREASED PITUITARY SIZE
- 32. **Measly red rope supporting large punching bag**: in pregnancy, vascular supply to the pituitary remains unchanged (despite increased pituitary size)
- 33. **Mother with bloody sheHANS**: SHEEHAN syndrome (ischemic necrosis of the pituitary in the postpartum period) can occur with acute blood loss during delivery (inadequate perfusion of the enlarged pituitary)
- 34. **Broken baby bottle** : in SHEEHAN syndrome, lactation is absent (HYPOpituitarism → decreased PROLACTIN production → decreased milk production)
- 35. White pants : in SHEEHAN syndrome, menses do not resume in the postpartum period (HYPOpituitarism \rightarrow decreased FSH and LH)
- 36. **Mother with antibody weapon**: LYMPHOCYTIC HYPOPHYSITIS (autoimmune disorder causing pituitary inflammation and necrosis) can cause HYPOpituitarism during late pregnancy or the postpartum period
- 37. Blue dot pattern: LYMPHOCYTIC HYPOPHYSITIS displays abundant cells with blue nuclei (due to lymphocyte infiltration)
- 38. **Skull within broken bag**: LYMPHOCYTIC HYPOPHYSITIS can cause pituitary necrosis and fibrosis → HYPOpituitarism
- 39. Moon face: LYMPHOCYTIC HYPOPHYSITIS is treated with corticosteroids
- 40. **Roof caving into punching bag**: PRIMARY EMPTY SELLA SYNDROME (anatomic defect with extension of dura into the sella → pressure atrophy of pituitary tissue → HYPOpituitarism)
- 41. Radiation skylight compressing punching bag : SECONDARY EMPTY SELLA SYNDROME (radiation, injury, or surgery → regression of pituitary gland → HYPOpituitarism)

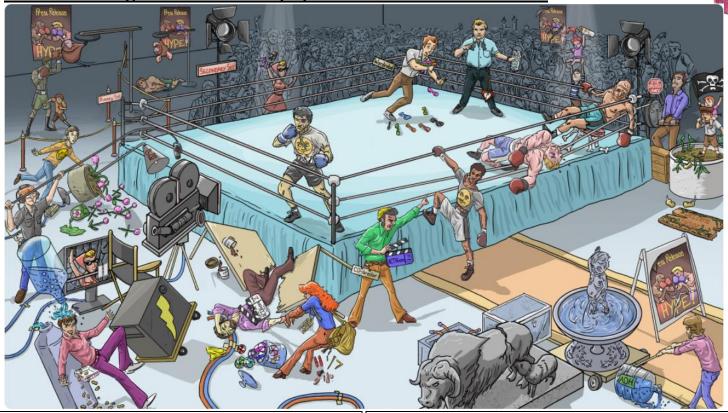
Endocrine 1.2 - Hypothalamic & Pituitary Dysfunction - Clinical Manifestations



- "HYPE" Press RELEASE: the HYPOTHALAMUS secretes RELEASING hormones → stimulates the ANTERIOR pituitary to secrete its own hormones
- 2. **FLAT PiG**: ANTERIOR pituitary secretes FSH, LH, ACTH, TSH, prolactin, growth hormone
- 3. **Punched in the gonads**: HYPOGONADISM is caused by deficiency in the GONADS (ovaries or testicles)
- 4. "**Primary SET**": in a PRIMARY (1º) endocrine disorder, the ENDOCRINE ORGAN is dysfunctional (gonads, thyroid gland, adrenal gland)
- 5. "HYPE" Press RELEASE: in 1º HYPOGONADISM, GONADOTROPIN RELEASING hormone (GnRH, secreted by HYPOTHALAMUS) is INCREASED (due to loss of negative feedback from gonadal hormones)
- 6. Elevating pituitary punching bag: in 1° HYPOGONADISM, LH and FSH (PITUITARY hormones) are INCREASED (due to loss of negative feedback from gonadal hormones and increased GnRH)
- 7. "Secondary SET" : in a SECONDARY (2°) endocrine disorder, the PITUITARY gland is dysfunctional
- 8. "HYPE" Press RELEASE: in 2º HYPOGONADISM, GONADOTROPIN RELEASING hormone (GnRH, secreted by HYPOTHALAMUS) is INCREASED due to loss of negative feedback from gonadal hormones)
- 9. Beat-up punching bag : $2^{\rm o}$ HYPOGONADISM is due to PITUITARY dysfunction (decreased LH and FSH)
- 10. **Fallen boxer**: in 2º HYPOGONADISM, GONADAL hormones are DECREASED (due to decreased stimulation by pituitary hormones [LH, FSH])
- 11. Falling FSH and LigHt with wilted gonadal flowers: 2° (HYPOgonadotropic) HYPOgonadism is caused by decreased LH and FSH release from the anterior pituitary (first sign of pituitary adenoma)
- 12. **Scattered female flowers**: in women, HYPOGONADISM leads to estrogen deficiency
- 13. **Paused video**: in women, HYPOGONADISM presents with premature ovarian failure (due to estrogen deficiency) and MENOPAUSAL symptoms (amenorrhea, infertility, vaginal atrophy, hot flashes, decreased breast tissue, decreased bone mineral density)
- 14. **Scattered male flowers**: in men, HYPOGONADISM leads to testosterone deficiency
- 15. Floppy boom mic: in men, HYPOGONADISM presents with impotence and infertility (due to TESTOSTERONE deficiency)

- 16. "X"d out shirt: in children, 2º HYPOGONADISM presents with absence of puberty (by age 14 in boys, age 12 in girls) and development of secondary sex characteristics (with LOW LH and FSH)
- 17. Falling "ACTHion" clapperboard and adrenal hat: in 2º ADRENAL INSUFFICIENCY, adrenal stimulation is decreased (because of decreased secretion of ACTH from the anterior pituitary)
- 18. **Ripped moon face**: in 2º ADRENAL INSUFFICIENCY, CORTISOL levels are decreased (due to decreased secretion of ACTH from the anterior pituitary)
- 19. Exhausted director: ADRENAL INSUFFICIENCY can cause weakness, fatigue, and muscle pain (due to decreased CORTISOL)
- 20. **Knocked over candy jar** : ADRENAL INSUFFICIENCY can cause hypoglycemia (decreased CORTISOL → decreased gluconeogenesis)
- 21. **Lightning bolt**: ADRENAL INSUFFICIENCY can cause SHOCK ("adrenal crisis") following a stressful event such as infection or surgery (unable to increase CORTISOL and maintain blood pressure)

Endocrine 1.2 - Hypothalamic & Pituitary Dysfunction - Clinical Manifestations



- 22. Intact CRYSTAL necklace: in 2º ADRENAL INSUFFICIENCY, MINERALocorticoid production by the adrenal gland is preserved (not affected by ACTH levels) → NO hypokalemia
- 23. **Untied bowtie**: HYPOTHYROIDISM is usually 1º (due to dysfunction of the thyroid gland), but can be 2º (due to pituitary dysfunction) or 3º (due to hypothalamic dysfunction)
- 24. Wet from water cooler: 2º ADRENAL INSUFFICIENCY can lead to elevated ANTIDIURETIC HORMONE (ADH) and free water reabsorption (low CORTISOL → hypotension → increased ADH)
- 25. Falling peanuts: 2º ADRENAL INSUFFICIENCY can cause hyponatremia (elevated ADH → increased water reabsorption)
- 26. **Makeup artist**: METYRAPONE blocks 11β-hydroxylase in the adrenal cortex (normally, administration leads to DECREASED CORTISOL → loss of negative feedback on anterior pituitary → INCREASED ACTH secretion
- 27. Unable to revive "ACTHIon" director: in 2º ADRENAL INSUFFICIENCY, METYRAPONE administration does NOT lead to increased ACTH secretion (because of anterior pituitary dysfunction)
- 28. Falling "11" lipstick: in 2º ADRENAL INSUFFICIENCY, serum 11-deoxycortisol (an intermediate in adrenal steroid synthesis) does NOT rise in response to METYRAPONE (due to low ACTH)
- 29. **Falling "17" makeup**: in 2º ADRENAL INSUFFICIENCY, urinary 17-hydroxycorticosteroid (an intermediate in adrenal steroid synthesis) does NOT rise in response to METYRAPONE (due to low ACTH)
- 30. **CO-director with plastic clapperboard** : COSYNTROPIN (synthetic ACTH) stimulates adrenal glands to secrete cortisol)
- 31. **Co-director activating moon-face**: in 2º ADRENAL INSUFFICIENCY, administration of COSYNTROPIN stimulates secretion of CORTISOL from the adrenal glands (in contrast to 1º insufficiency, where cortisol is NOT produced regardless of adrenal stimulation)
- 32. Obese ref with ice cup & falling heart watch: HYPOTHYROIDISM presents with weight gain, cold intolerance, bradycardia, constipation, slow deep tendon reflexes
- 33. Falling "bowtie stimulating" wardrobe guy: in 2º HYPOTHYROIDISM, TSH (and THYROID hormone) is LOW
- 34. Falling bean sprouts: GROWTH HORMONE (GH) DEFICIENCY (due to anterior pituitary dysfunction)
- 35. **Short kid**: in CHILDREN, GH DEFICIENCY presents with growth failure

- 36. **Infantile kid**: in children, GH DEFICIENCY can present with immature face, infantile "cherub-like" fat distribution, decreased hair growth, small genitalia and delayed puberty
- 37. Younger kid holding skull & crossbones : in children, GH DEFICIENCY presents with delayed bone age (bone age < chronological age) (measured using X-rays)
- 38. **Obese out-of-shape boxer**: in adults, GH DEFICIENCY presents with muscle wasting and truncal obesity
- 39. **Breaking bone stool**: in adults, GH DEFICIENCY can cause decreased bone density and osteoporosis
- 40. Vines GROWing on "Welcome INSide": insulin-like growth factor 1 (IGF-1) is LOW in GH DEFICIENCY (normally secreted by liver in response to GH)
- 41. "L" & "R" rope crank : administration of L-dopa and aRginine normally leads to increased GH (L-dopa and arginine → decreased somatostatin secreted by GI system → decreased inhibition on anterior pituitary → increased GH secretion)
- 42. Pooped-out despite cranking to the max: in GH DEFICIENCY, administration of L-DOPA and aRginine does NOT increase GH secretion (because pituitary is dysfunctional regardless of somatostatin level)
- 43. Raised baby bottle with torn "HYPE" press release:
 HYPOTHALAMIC dysfunction leads to INCREASED PROLACTIN levels
 (decreased DOPAMINE release from hypothalamus → decreased inhibition of prolactin secretion from anterior pituitary)
- 44. **Ponytail STALK**: severing or injuring PITUITARY STALK leads to INCREASED PROLACTIN levels (decreased DOPAMINE reaching anterior pituitary → decreased inhibition of prolactin secretion)
- 45. **Broken OX feeding calf**: in hypothalamic dysfunction, decreased OXYTOCIN from the POSTERIOR pituitary can decrease uterine contractions during childbirth and milk letdown in breastfeeding
- 46. Insipidus fountain and falling ADH bottle: in hypothalamic dysfunction, decreased ADH from the POSTERIOR pituitary causes DIABETES INSIPIDUS (DI) (inappropriately dilute urine, polyuria, hypernatremia)
- 47. **Torn "HYPE" press release**: oxytocin and ADH deficiency (DI) are more common with hypothalamic injury (NOT posterior pituitary injury) because these hormones are made in the hypothalamus

Endocrine 1.3 - Hyperpituitarism





- 1. #1 grandPaPPy: pituitary adenoma formation is one of the features of Multiple Endocrine Neoplasia type I (Pituitary, Pancreas, Parathyroid)
- 2. **McCune mosaic puzzle** : McCune-Albright displays genetic mosaicism, in which mutation occurs AFTER fertilization→ mixture of mutated and normal genotypic cells
- 3. **G lamp over cAMP story**: McCune albright is due to a mutation in G-protein signaling→ increased cAMP→ overactive endocrine tissue (including pituitary)
- 4. **Coffee stains on one page**: unilateral cafe-au-lait macules are a component of the triad of findings in McCune-Albright syndrome
- 5. "Precocious learner!": pituitary hyperfunction in McCune-Albright syndrome can lead to excess LH and FSH (precocious puberty) or growth hormone (gigantism)
- 6. **Fibrous bone plant** : polyostotic fibrous dysplasia (replacement of bone and marrow with fibrous tissue→ bone pain and fractures) is a component of the McCune-Albright triad
- 7. One PL bottle: prolactinoma (adenomatous growth of lactotroph cells) is the most common functional pituitary adenoma
- 8. PL bottles tearing GardneR's Hardware bag : prolactin inhibits the release of GnRH from the hypothalamus→ decreased LH and FSH
- Knocked over FSH bottle and LH lamp: inhibition of GnRH release from hyperprolactinemia→ decreased LH and FSH→ decreased sex hormones→ hypogonadism
- White skirt on mom with PL bottles: hypogonadism (secondary to hyperprolactinemia) in premenopausal women with prolactinomas causes amenorrhea
- 11. "The Barren Tree" uterus: hypogonadism (secondary to hyperprolactinemia) in premenopausal women with prolactinomas causes infertility
- 12. "The Impotence Bride" floppy sword: hypogonadism (secondary to hyperprolactinemia) in men with prolactinomas causes impotence
- 13. Boy squirting milk: hypogonadism (secondary to hyperprolactinemia) in men with prolactinomas can RARELY cause galactorrhea
- 14. Baby discharging milk onto mom's shirt: hyperprolactinemia in premenopausal women causes milky nipple discharge (galactorrhea)
- 15. Cabergoline burglar with bromocriptine broom handle bindle: cabergoline and bromocriptine (D2 dopamine agonists) inhibit prolactin production by lactotrophs (pharmacologic treatment of prolactinoma)
- 16. **D2 double dopamine rope**: D2 dopamine agonists (cabergoline, bromocriptine) inhibit prolactin production by lactotrophs (pharmacologic treatment of prolactinoma)
- 17. Growth hormone bean stalks :growth hormone-producing adenomas (GHomas) are the second most common functional pituitary adenomaGrowth hormone bean stalks : growth hormone-producing adenomas (GHomas) are the second most common functional pituitary adenoma
- 18. Welcome Inside sign growing through liver shaped hole: growth hormone stimulates the liver the produce insulin-like growth factor-1 (IGF-1) → elevated IGF-1 levels with GHoma
- 19. **Tall, skinny young giant**: in children, GHoma→ excess GH and IGF-1→ rapid LINEAR growth before closure of epiphyseal plates (extremely tall stature)→ pituitary gigantism
- 20. **Stocky, short older giant**: in adults, GHoma→ excess GH and IGF-1→ NON-LINEAR bone growth (frontal bossing, macrognathia, enlarged hands and feet)→ acromegaly

- 21. Apneic blue giant with protruding tongue: excess growth hormone in acromegaly causes soft tissue enlargement → macroglossia, upper airway obstruction, and sleep apnea
- 22. **Giant coarse beard**: women with GH excess in acromegaly may show signs of masculinization (hirsutism), such as facial hair growth
- 23. **Giant wrapped joints**: GH excess in acromegaly can cause hypertrophy and stiffening of synovial tissue→ hypertrophic arthropathy
- 24. **Giant organ shirt**: GH excess in acromegaly can cause visceromegaly (especially of the heart, liver, and thyroid)
- 25. Giant polyp garden: patients with acromegaly have an increased incidence of colon polyps and cancer
- 26. Giant pressure steam : increased GH and insulin levels in acromegaly cause sodium and volume retention \rightarrow hypertension
- 27. **Giant floppy heart balloon**: hypertension and left ventricular hypertrophy in acromegaly often lead to heart failure, arrhythmia, and valve disease (especially aortic regurgitation)
- 28. **Giant candy jar** : growth hormone stimulates gluconeogenesis and causes hyperglycemia→ insulin resistance, elevated insulin levels, and type II diabetes mellitus
- 29. Raised candy and GH bean: administration of oral glucose (glucose suppression test) will NOT lower GH levels in a patient with a GH producing
- 30. Cabergoline burglar by stop sign and giant ants: cabergoline (D2 dopamine agonist), pegvisomant (GH receptor antagonist), and octreotide (somatostatin analog) are all part of pharmacologic GHoma treatment
- 31. Baby giant with PL bottle and X-eyes shirt: in children (pituitary gigantism), GHomas are made up of mammosomatotroph cells, which secrete BOTH prolactin and GH→ pituitary gigantism AND hyperprolactinemia (delayed puberty, lack of secondary sex characteristics)
- 32. **Tightening big red bowtie**: pituitary adenomas may produce TSH (thyrotroph adenoma) → hyperthyroidism (though most cases do NOT cause hyperthyroidism due to dysfunctional TSH production)
- 33. 4 pattern on bow tie: hyperthyroidism due to a TSH-producing pituitary adenoma (secondary hyperthyroidism) will show both a HIGH T4 and HIGH TSH (opposed to high T4 and LOW TSH in primary hyperthyroidism)
- 34. **Fat bowtie knot** : chronic TSH secretion from a pituitary adenoma causes thyroid hypertrophy and hyperplasia→ goiter formation
- 35. **Nervous sweaty kid**: secondary hyperthyroidism caused by TSH-producing pituitary adenomas has the same features of primary hyperthyroidism (sweating, weight loss, tremor, palpitations)
- 36. **LH lamp and FSH bottle on the floor**: gonadotrophic pituitary adenomas are almost alway NON-functional and only cause symptoms due to mass effect (headache, visual changes)
- 37. **ACTH ACTION clapper** : ACTH-producing pituitary adenomas caused chronic stimulation of the adrenal cortex→ increased production of cortisol
- 38. **Eerie moon face** : chronic stimulation of the adrenal cortex due to an ACTH-producing adenoma→ hypercortisolism
- 39. Large cushion : ACTH-producing adenoma→ hypercortisolism→ fat redistribution, thin skin, obesity, insulin resistance (Cushing's DISEASE)

Endocrine 1.4 - Diabetes Insipidus & SIADH



- 1. **Water backpack**: the POSTERIOR pituitary secretes antidiuretic hormone (ADH) (vasopressin) (promotes free water conservation)
- 2. **Ox feeding calf**: the POSTERIOR pituitary secretes OXYTOCIN, important in labor (responsible for uterine smooth muscle contractions) and lactation (stimulates milk letdown by surrounding)
- 3. Nucleus-like transformer adjacent to VENT & OCULAR security camera: OXYTOCIN and ADH are made in the paraVENTRICULAR and supraOCULAR nuclei in the HYPOTHALAMUS
- 4. Wires traveling down from nucleus transformer : OXYTOCIN and ADH are transported down axons from the HYPOTHALAMIC nuclei to the POSTERIOR PITUITARY → stored in nerve terminals (therefore they can be secreted even if the posterior pituitary is injured)
- 5. **RED Ketchup condOSMent cart**: ADH plays a role in maintaining PLASMA OSMolality (plasma solutes/amount of solvent) (a concentration)
- 6. **SALTY peanuts**: OSMolality is derived primarily from serum SODIUM levels (serum GLUCOSE and BUN levels also contribute)
- 7. "3Cs" logo on RED condOSMent cart : SERUM OSMolality levels above 300 are elevated (normal range: 275-295 mOsm/Kg)
- 8. Raised red ketchup condOSMents : PLASMA HYPERosmolality stimulates osmoreceptors in the hypothalamus \rightarrow triggers posterior pituitary to secrete ADH
- 9. **Empty cups**: significant HYPOVOLEMIA stimulates ADH secretion by the posterior pituitary (dehydration, blood loss, cirrhosis, heart failure)
- 10. ["V" V" sign: V1 and V2 are the main peripheral ADH receptors
- 11. **["Q"** : V1 receptors activate Gq proteins located in VASCULAR SMOOTH MUSCLE (constriction)
- 12. **Tight red straps**: stimulation of V1 receptors cause VASOCONSTRICTION (\rightarrow increased blood pressure) (Gq increases intracellular calcium \rightarrow vascular constriction)
- 13. **["S"**: V2 receptors activate Gs proteins and are located in the KIDNEY (principal cells of the collecting tubules)
- 14. **Abundant AQP waters**: stimulation of V2 receptors → activates Gs → upregulates the expression of AQUAPORIN channels → INCREASED REABSORPTION OF FREE WATER (at the COLLECTING DUCT)
- 15. **Broken ADH with brain hat**: CENTRAL DIABETES INSIPIDUS (a result of decreased ADH production by the CNS (hypothalamus))
- 16. **Saying "no" to water peddler**: NEPHROGENIC DIABETES INSIPIDUS (caused by resistance to ADH in the kidneys) (either secondary to insufficient number of ADH receptors OR dysfunctional aquaporins)

- 17. Full ADH backpack: in NEPHROGENIC DIABETES INSIPIDUS, ADH levels are normal (due to fully functional CNS)
- 18. Fountain of dilute urine: DIABETES INSIPIDUS (DI)
- 19. **Dilute "CCC" fountain**: in DI, URINE OSMolality levels are LOW (< 300 mOsm/kg)
- 20. Generous pee stream: DI presents with POLYURIA
- 21. **Elevated salty peanuts**: DI presents with HYPERnatremia (serum sodium > 145) and increased serum OSMolality (> 290)
- 22. **Drinking large stream of water** : DI presents with POLYDIPSIA (increased thirst) \rightarrow increased water intake helps minimize serum osmolality increase
- 23. **Old man and baby with peanuts**: in DI, the elderly and infants are at greatest risk for HYPERNATREMIA (due to inability to increase fluid intake to compensate for free water loss)
- 24. ["Idiota": CENTRAL DI is commonly IDIOPATHIC
- 25. **Antibody trident**: AUTOIMMUNE disease can cause CENTRAL DI (may underlie many "idiopathic" cases) (may destroy pituitary stalk and body)
- 26. **Crab crown**: a brain tumor affecting the HYPOTHALAMUS can cause CENTRAL DI (however, an isolated PITUITARY tumor will usually NOT cause DI because ADH is made in the hypothalamus)
- 27. **Scalpel and head cracks**: surgery in the hypothalamic and pituitary regions can cause CENTRAL DI
- 28. $\ensuremath{\mathbf{Li}}$ balloons : LITHIUM therapy is the most common cause of NEPHROGENIC DI
- 29. **Knocking over AQP bottles**: LITHIUM prevents proper aquaporin formation (within principal cells in the collecting duct)
- 30. **Raising calci-YUM ice cream**: HYPERCALCEMIA can cause NEPHROGENIC DI (may be due to decreased aquaporin channels, direct osmotic effect, and inhibition of salt reabsorption in the loop of Henle)
- 31. **Kid resisting AQP water**: hereditary conditions can cause NEPHROGENIC DI (mutations in genes encoding V2 receptors or aquaporins)
- 32. 'Do Not Drink": a water-deprivation test can be used to differentiate between DI and primary polydipsia (drinking excessive volumes of water)
- 33. Concentrated condOSMents in water: a water-deprivation test will lead to INCREASED URINE osmolality if ADH is fully functional (e.g. primary polydipsia) (due to increased free water reabsorption)

Endocrine 1.4 - Diabetes Insipidus & SIADH



- 34. Raising peanuts while losing water: a water-deprivation test in DI will INCREASE SERUM osmolality (>295 mOsm/kg) (due to inability to increase free water absorption)
- 35. **Dilute water leak**: in DI, a water-deprivation test will NOT raise URINe OSMolality (stays LOW <300 mOsm/kg) (due to inability to increase free water absorption)
- 36. **Refilling ADH**: desmopressin (an ADH-analog) can be administered to distinguish between CENTRAL and NEPHROGENIC DI
- 37. **Brain cap kid peeing on condOSMents**: in CENTRAL DI, administration of desmopressin will INCREASE URINE osmolality (exogenous ADH-analog is acting on NORMAL kidneys) (nephrogenic DI will show no response)
- 38. **Replacing ADH backpack**: CENTRAL DI is treated with exogenous ADH-analogs
- 39. **Chloro-thighs**: NEPHROGENIC DI can be treated with THIAZIDE DIURETICs (increases volume depletion → upregulates volume conservation in proximal nephron)
- 40. **Almonds**: NEPHROGENIC DI can be treated with amiloride (potassium sparing diuretic) (increases volume depletion → upregulates volume conservation in proximal nephron)
- 41. Anti-inflammatory fire extinguisher : NEPHROGENIC DI can be treated with NSAIDS (\rightarrow decreased prostaglandins \rightarrow decreased inhibition of ADH)
- 42. **['UNSALTED"** : NEPHROGENIC DI can be treated with a low-salt low-protein diet
- 43. **Inappropriate ADH water from head**: syndrome of inappropriate ADH (SIADH) (excessive ADH secretion)
- 44. **Diluted peanuts**: SIADH presents with HYPOnatremia (sodium < 135) (due to excessive reabsorption of free water)
- 45. "Euvolemic" bucket next to spilled Peanuts: SIADH presents with Euvolemic Hyponatremia
- 46. Large wet kid: in SIADH, initially volume increases (due to increased ADH and excessive free water retention)
- 47. **BNP blimp**: in SIADH, the atria and ventricles release AMP and BNP (due to detection of initial volume overload)
- 48. Throwing peanuts into fountain: in SIADH, the kidney excretes additional sodium (as a result of increased ANP and BNP)

- 49. **Discarding RAIN umbrella**: in SIADH, the renin angiotensin aldosterone system (RAAS) is DEACTIVATED (due to detection of volume expansion) → DECREASED aldosterone
- 50. **Dropping even MORE peanuts**: in SIADH, the kidney excretes additional sodium (as a result of RAAS deactivation)
- 51. **Bucket to "Euvolemic" middle fill line**: SIADH presents with EUVOLEMIC hyponatremia (initial H2O retention --> +ADH, -RAAS --> vol excretion)
- 52. "100 euros": SIADH presents with urine osmolality >100 mOsm/kg (inappropriately CONCENTRATED urine)
- 53. **Peanuts floating in watery fountain**: SIADH presents with ELEVATED URINE SODIUM (> 40 mEq/L) (due to increased sodium excretion [a result of increased BNP and decreased aldosterone])
- 54. Stroke of black: Stroke and meningitis can cause SIADH
- 55. Cracked head: Head trauma can cause SIADH
- 56. Crab breast plate : small cell lung cancer can cause SIADH
- 57. **Scalpel staff**: surgery can cause SIADH (pain, inflammation, or direct pituitary injury → increased secretion of ADH)
- 58. **Mortar & pestle** : drugs (carbamazepine, SSRIs, cyclophosphamide) can cause SIADH
- 59. Cyclops: cyclophosphamide can cause SIADH
- 60. "Restricted water access": mild SIADH can be treated with free water restriction
- 61. Peanuts: SIADH can be treated with salt administration
- 62. **IVY over peanut-saturated jail cell**: severe SIADH can be treated with IV HYPERtonic saline
- 63. "VAPorizer": severe SIADH can be treated with V2 receptor blockers (CONIVAPTAN, TOLVAPTAN) (directly inhibit action of ADH)
- 64. 6BiCYCLE: severe SIADH can be treated with DEMECLOCYCLINE (acts at collecting tubule to block ADH signaling)

Endocrine 2.1 - Adrenal Insufficiency





- 1. **Mineral crystal on top**: mineralocorticoids (aldosterone) are made in the zona glomerulosa (the outer layer of the adrenal cortex)
- 2. **Moon face in the middle** : glucocorticoids (cortisol) is made in the zona fasciculata (the middle layer of the adrenal cortex)
- 3. **Male symbol on the bottom**: androgens are made in the zona reticularis (the innermost layer of the adrenal cortex)
- 4. Cracked Cortical Studios arch : adrenal insufficiency (can be 1° [intrinsic defect], 2° [low ACTH], or 3° [low CRH])
- 5. Set 1 : primary adrenal insufficiency (caused by intrinsic defect of the adrenal cortex)
- 6. **Bleeding adrenal beanies**: bilateral adrenal hemorrhage can cause acute 1° adrenal insufficiency (e.g. due to anticoagulation or DIC)
- 7. **WF water tower**: Waterhouse-Friderichsen syndrome (bilateral adrenal hemorrhage in acute sepsis)
- 8. **Septic sewer plate**: Waterhouse-Friderichsen often occurs in the setting of sepsis and septic shock (e.g. due to Neisseria meningitidis)
- 9. **Broken moon face**: loss of cortisol production (hypocortisolism main component of adrenal crisis)
- 10. **Dilated red arterial sleeves** : lack of cortisol→ decreased vascular tone→ hypotension
- 11. Fainting with lightning bolts : cortisol deficiency→ hypotension and shock
- 12. **Falling candies** : cortisol deficiency→ decreased gluconeogenesis→ hypoglycemia
- 13. **Broken mineral crystals** : 1° adrenal insufficiency \rightarrow decreased mineralocorticoid production (hypoaldosteronism)
- 14. **Raised bananas**: hypoaldosteronism→ decreased Na/K counter transporter activity in the collecting ducts→ HYPERkalemia (1° adrenal insufficiency)
- 15. **4 acid tubes** : hypoaldosteronism→ type 4 renal tubular acidosis (due to decreased ammonia production) (1° adrenal insufficiency)
- 16. Falling salty peaNa+s: 1° adrenal insufficiency is associated with hyponatremia (due to increased ADH secretion)
- 17. Falling water and peaNa+s : hypoaldosteronism→ increased renal excretion of sodium AND water→ volume loss
- 18. Falling salty peaNa+s: 1* adrenal insufficiency is associated with hyponatremia (due to increased ADH and free water reabsorption)
- 19. **Elevated ADH water cooler** : hypoaldosteronism→ hypovolemia→ ADH release→ increased free water absorption→ hyponatremia (due to dilution of the solutes in the serum)
- 20. Daisy Addison: Addison's disease (Chronic 1° adrenal insufficiency)
- 21. Chronic grandfather clock : Addison's disease follows a chronic course with insidious onset
- 22. Daisy Addison's antibody chair: Addison's disease is caused by autoimmune destruction of the adrenal cortex
- 23. Hollywood B8ul3v4rd Los Angeles : Addison's disease is associated with HLA B8, DR3, and DR4

- 24. **Pulmonary cacti with cavitary lesions** : disseminated tuberculosis can cause CHRONIC 1* adrenal insufficiency
- 25. Sitting up and passed out : postural hypotension (low cortisol \rightarrow decreased vascular tone) (Addison's disease)
- 26. **Spilled drink** : hypoaldosteronism \rightarrow salt wasting \rightarrow volume loss (Addison's disease)
- 27. **Spray tan**: Addison's disease is associated with skin hyperpigmentation (e.g. oral mucosa, face, hands, genitalia)
- 28. Raised ACTHion clapperboard : 1° adrenal insufficiency→ decreased cortisol→ increased ACTH (due to loss of negative feedback from cortisol)
- 29. **MSH spray tan** : MSH increases as a result of increased ACTH production \rightarrow hyperpigmentation
- 30. White skirt: Addison's disease can cause irregular menses or amenorrhea
- 31. **Elelvated ACTHion clapperboard**: ACTH is increased in 1° adrenal insufficiency
- 32. Set ${\bf 2}$: secondary adrenal insufficiency (due to decreased ACTH from pituitary)
- 33. **Pituitary punching bag** : 2° adrenal insufficiency is due to a disorder of the pituitary gland (\rightarrow decreased ACTH \rightarrow decreased cortisol)
- 34. **Broken ACTHion clapperboard**: 2° adrenal insufficiency causes decreased ACTH (due to pituitary dysfunction)
- 35. Dropping MSH spray tan : MSH levels are low in 2° adrenal insufficiency (because ACTH is not being made)
- 36. Preserved mineral crystals : mineralocorticoid production is not affected by decreased ACTH in 2° adrenal insufficiency
- 37. Stuffing falling from pituitary punching bag : 2° adrenal insufficiency is often associated with other hormone deficiencies (e.g. LH, FSH, and TSH)
- 38. **Set 3**: tertiary adrenal insufficiency (due to lack of CRH release from the hypothalamus) (tumor, radiation, exogenous steroids, etc.)
- 39. **Broken produCRH chair**: decreased conticotropin releasing hormone (CRH) due to hypothalamic inhibition (3° adrenal insufficiency)
- 40. **Moon face crushing produCRH**: chronic steroid therapy is the most common cause of 3° adrenal insufficiency (suppress CRH release from hypothalamus)
- 41. **Broken ACTHion clapperboard** : chronic exogenous steroids→ decreased CRH→ decreased ACTH → decrease endogenous cortisol
- 42. Fallen adrenal beanie : chronic exogenous steroids \to decreased CRH \to decreased ACTH \to adrenal ATROPHY
- 43. **Preserved mineral crystals**: mineralocorticoid (aldosterone) production is NOT affected by decreased ACTH in 3* adrenal insufficiency (controlled by RAAS)
- 44. Passed out behind moon : acutely stopping exogenous steroids \rightarrow low endogenous cortisol unmasked \rightarrow adrenal insufficiency/crisis
- 45. Long tapering moon flag : to avoid adrenal insufficiency, taper off steroid therapy
- 46. "Infection...Injury...Crisis": acute stress events (e.g. injury, surgery, infection) increase body's demand for endogenous cortisol \rightarrow adrenal crisis in patients with 1°, 2°, 3° adrenal insufficiency (e.g. those on chronic steroids not adequately dosed))

Endocrine 2.2 - Congenital Adrenal Hyperplasia (CAH)



- 1. MINERAL crystals in GEM Cave: zona GLOMERULOSA (site of MINERALocorticoid synthesis) (outer layer of adrenal cortex)
- 2. MOON-faced fruit in FASCICULAR forest : zona FASCICULATA (site of GLUCOCORTICOID synthesis) (middle layer of adrenal cortex)
- 3. AIRship: zona RETICULARIS (site of ANDROGEN synthesis) (inner layer of adrenal cortex)
- 4. Adrenal hat with ACTHion clapper board: adrenocorticotropic hormone (ACTH) (secreted by anterior pituitary) stimulates the adrenal cortex to begin hormone production
- 5. **Jumping "Super DESMOND"**: ACTH upregulates the enzyme DESMOLASE → converts cholesterol to pregnenolone (rate-liming step of steroid hormone synthesis)
- 6. Cholesterol coins : CHOLESTEROL is converted to pregnenolone (via desmolase)
- 7. **PIRanha plant**: PREGnenolone (starting molecule for all hormones synthesized by the adrenal cortex) is synthesized from cholesterol (via desmolase)
- 8. **Turtle with PROtective dome**: PROgesterone (synthesized from pregnenolone via 3-beta-hydroxysteroid dehydrogenase)
- 9. **Monkey with "21" tail & tie** : 21-hydroxylase (converts progesterone to 11-deoxycorticosterone)
- 10. **Blue STONY turtle with "11" suspenders**: 11-deoxycorticoSTERONE (synthesized from progesterone via 21-hydroxylase)
- 11. "11" suspenders : 11-hydroxylase (converts 11-deoxycorticosterone to corticosterone)
- 12. **STONY turtle**: corticoSTERONE (synthesized from 11-deoxycorticosterone via 11-hydroxylase)
- 13. Lizard with MINERAL shell: ALDOSTERONE (synthesized from corticosterone)
- 14. **Tense red bands**: ANGIOTENSIN 2 (product of renin-angiotensin cascade) is required for synthesis of aldosterone)
- 15. "18" sword & shield : 18-hydroxylase (converts corticosterone to aldosterone) is activated by angiotensin 2
- 16. **Teleport piper**: 17-hydroxylase (converts pregnenolone to 17-hydroxypregnenolone)

- 17. **PIRanha plant with "17" sunglasses**: 17-hydroxyPREGnenolone (synthesized from pregnenolone via 17-hydroxylase)
- 18. **Turtle with PROtective dome & "17" sunglasses**: 17-hydroxyPROgesterone (synthesized from 17-hydroxypregnenolone by 3-beta-hydroxysteroid dehydrogenase)
- 19. **Teleport pipe**: 17-hydroxylase (converts progesterone to 17-hydroxypregnenolone)
- 20. **Monkey with "21" tail & tie**: 21-HYDROXYLASE (converts 17-hydroxyprogesterone to 11-deoxycortisol)
- 21. **Blue MOON turtle with "11" suspenders**: 11-deoxyCORTISOL (synthesized from 17-hydroxyprogesterone via 21-hydroxylase)
- 22. **Moon wizard**: CORTISOL (synthesized from 11-deoxyCORTISOL via 11-hydroxylase)
- 23. **Brother with "11" suspenders**: 11-HYDROXYLASE (converts 11-deoxyCORTISOL to CORTISOL)
- 24. **Teleport pipe**: 17-hydroxylase (converts 17-hydroxypregnenolone to DHEA)
- 25. **DEHYDRATED bones throwing ANDROGEN hammers**: DEHYDROepiANDROSTERONE (DHEA) (synthesized from 7-hydroxypregnenolone via 17-hydroxylase)
- 26. **TWO turtles throwing ANDROGEN hammers**: ANDROsteneDIONE (synthesized from DHEA via 3-beta-hydroxysteroid dehydrogenase)
- 27. "17" KEYS: 17-KETOsteroids (alternative term for DHEA & androstenedione) ("weak" androgens)
- 28. **BIG turtle throwing ANDROGEN hammers**: TESTOSTERONE (synthesized from androstenedione via oxidoreductase)
- 29. **Hiding in pipe**: 11, 17, and 21 hydroxylase deficiencies are autosomal recessive
- 30. Large adrenal hat and jumping with ACTHion: CAH leads to adrenal gland hyperplasia (decreased cortisol synthesis \rightarrow negative feedback on anterior pituitary \rightarrow increased ACTH \rightarrow overgrowth of adrenal gland
- 31. **Adding black paint**: CAH leads to hyperpigmentation (because MSH increases along with ACTH [POM-C is cleaved into ACTH and MSH])

Endocrine 2.2 - Congenital Adrenal Hyperplasia (CAH)



- 32. **Princess sitting on "?"**: in females, severe 11 and 21-hydroxylase deficiency can present at birth with ambiguous genitalia (due to excessive androgen exposure in utero)
- 33. **Enlarged nose**: in females, mild 11 and 21-hydroxylase deficiency can present in adolescence with clitoromegaly (due to HYPERandrogenism)
- 34. **Early moustache**: in females, mild 11 and 21-hydroxylase deficiency can present in adolescence with precocious pubic hair development or hirsutism (due to HYPERandrogenism)
- 35. **Irregular red coins**: in females, 11 and 21-hydroxylase deficiency can present with irregular menses and infertility (due to HYPERandrogenism)
- 36. **Broken mineral with "21" monkey**: 21-hydroxylase deficiency leads to decreased aldosterone
- 37. Chomped moon fruit with "21" monkey: 21-hydroxylase deficiency leads to decreased cortisol
- 38. **Falling peanuts**: 21-hydroxylase deficiency leads to HYPOnatremia (due to HYPOaldosteronism) ("salt wasting")
- 39. **Raised bananas**: 21-hydroxylase deficiency leads to HYPERkalemia (due to HYPOaldosteronism)
- 40. **Erupting yellow volcano**: 21-hydroxylase deficiency leads to type IV renal tubular ACIDOSIS (due to HYPOaldosteronism
- 41. **Fainting "21" monkey**: 21-hydroxylase deficiency can present with hypotension (due to HYPOaldosteronism and HYPOcortisolism)
- 42. "21" monkey with lightning bolt tail: 21-hydroxylase deficiency can present with hypovolemic shock (due to HYPOaldosteronism and HYPOcortisolism)
- 43. **Raised RAIN umbrella**: 21-hydroxylase deficiency leads to increased RENIN (decreased aldosterone → hypovolemia → increased renin angiotensin cascade)
- 44. **Broken mineral with "11" brother** : 11-hydroxylase deficiency leads to DECREASED aldosterone
- 45. **Pointing UP to "11" blue STONEY turtle** : 11-hydroxylase deficiency leads to increased 11-deoxycorticoSTERONE (exerts mineralocorticoid effects \rightarrow mimics HYPERaldosteronism)

- 46. **Steaming angry**: 11-hydroxylase deficiency leads to hypertension (due to increased mineralocorticoid effects) ("salt retainers")
- 47. **Falling bananas**: 11-hydroxylase deficiency leads to HYPOkalemia (due to increased mineralocorticoid effects)
- 48. **Falling RAIN umbrella**: 11-hydroxylase deficiency leads to decreased RENIN (due to increased mineralocorticoid effects)
- 49. Sunlight on "17" turtle with PROtective dome: 17-hydroxyPROgesterone builds up in both 11 and 21-hydroxylase deficiencies
- 50. **Sunlight on "11" blue STONEY turtle**: 11-deoxycorticoSTERONE builds up in 11-hydroxylase deficiency
- 51. Blocked by stone stomper with "17" sunglasses: 17-hydroxylase deficiency
- 52. **Covered with mineral crystals**: 17-hydroxylase deficiency leads to increased aldosterone (and decreased cortisol & androgens)
- 53. **Angry stomper**: 17-hydroxylase deficiency presents with hypertension (and other signs of excessive mineralocorticoids)
- 54. **2 cracked eggs**: 17-hydroxylase deficiency leads to severely decreased androgen production (because 17-hydroxylase is required for androgen synthesis in the gonads)
- 55. **Male dino sitting on "?"**: in males, 17-hydroxylase deficiency presents at birth with ambiguous (or female-appearing) genitalia (due to decreased testosterone)
- 56. **Crossed-out smiley face**: in females, 17-hydroxylase deficiency presents with decreased secondary sex characteristics (decreased androgens → decreased estrogen → no breast development) (decreased androgens → minimal body hair)
- 57. White underbelly: in females, 17-hydroxylase deficiency presents with delayed menarche or amenorrhea (due to decreased estrogen)

Endocrine 2.3 - Adrenocortical Hyperfunction: Hyperaldosteronism & Hypercortisolism



- 1. **Giant MINERAL crystals**: PRIMARY HYPERALDOSTERONISM (a MINERALOCORTICOID secreted by the zona GLOMERULOSA) (hypersecretion of aldosterone due to ADRENAL gland dysfunction)
- 2. "**Idiot**": bilateral IDIOPATHIC HYPERALDOSTERONISM (the most common cause of PRIMARY HYPERALDOSTERONISM)
- 3. **Overgrowing adrenal hat**: in bilateral IDIOPATHIC HYPERALDOSTERONISM, the zona GRANULOSA of both adrenal glands is HYPERPLASTIC
- 4. Well-circumscribed "ADDition" light on adrenal cap:
 ALDOSTERONE-secreting ADENOMA (unilateral, solitary, yellow, well circumscribed tumor) (2nd most common cause of PRIMARY HYPERALDOSTERONISM)
- 5. **Light turned-on**: ALDOSTERONE-secreting ADENOMAs produce hormone (aldosterone)
- 6. **Pink spots on SPIRAL notebook**: ALDOSTERONE-secreting ADENOMAS display "SPIRONOLACTONE bodies" (eosinophilic cytoplasmic inclusions)
- 7. **Normal 2nd adrenal hat**: ALDOSTERONE-secreting ADENOMAS do NOT cause atrophy of the contralateral adrenal gland (because ACTH secretion is not affected [ACTH is responsible for adrenal gland size])
- 8. **Creeping crab**: ADRENAL GLAND CARCINOMA & ECTOPIC ALDOSTERONE-secreting TUMORS (kidney, ovaries) can cause PRIMARY HYPERALDOSTERONISM
- 9. High pressure geyser: HYPERALDOSTERONISM causes SECONDARY HYPERTENSION (usually severe) (upregulation of renal sodium-potassium pumps \rightarrow sodium retention \rightarrow volume retention \rightarrow hypertension)
- 10. **Falling bananas**: HYPERALDOSTERONISM causes HYPOKALEMIA (upregulation of renal sodium-potassium pumps → excretion of potassium in the urine)
- 11. **Alkalotic blue mineral pools**: HYPERALDOSTERONISM causes metabolic ALKALOSIS (secondary to HYPOKALEMIA) (due to renal K+/H+ exchange, activation of renal H+atpases, K+/H+ exchange at cellular membrane)
- 12. **Escaping airship**: PRIMARY HYPERALDOSTERONISM activates the "aldosterone escape" mechanism (leads to some improvement in volume overload and hypernatremia)
- 13. Overloaded steam engine tank: PRIMARY HYPERALDOSTERONISM initially causes severe volume overload and hypertension (due to sodium and water retention)

- 14. Fluid burst streaming through glomerular pipe: PRIMARY HYPERALDOSTERONISM leads to INCREASED renal blood flow (RBF) and INCREASED glomerular filtration rate (GFR) (due to arterial hypertension)
- 15. Pressure promoting loss of water and peanuts: PRIMARY HYPERALDOSTERONISM eventually leads to "pressure natriuresis" (INCREASED EXCRETION of SODIUM and WATER) (due to increased GFR)
- 16. **Tensely inflated balloons**: PRIMARY HYPERALDOSTERONISM activates stretch receptors in the atrium (due to volume overload)
- 17. "ANP" flag coming: PRIMARY HYPERALDOSTERONISM leads to release of atrial natriuretic peptide (ANP) from the atria (due to activation of stress receptors) (leads to INCREASED SODIUM and WATER EXCRETION at the collecting duct)
- 18. **Well-balanced peanuts & water tank**: In PRIMARY HYPERALDOSTERONISM, the "aldosterone escape" mechanism leads to a steady state of MILD HYPERNATREMIA and MILD HYPERVOLEMIA
- 19. **Dropping closed umbrella**: PRIMARY HYPERALDOSTERONISM presents with LOW RENIN levels (due to increased blood pressure)
- 20. Excessive peanut consumption: administration of salt supplements can be used to diagnose PRIMARY HYPERALDOSTERONISM ("salt suppression test")
- 21. **Frightened by geyser near large minerals**: in PRIMARY HYPERALDOSTERONISM, a "SALT SUPPRESSION" test demonstrates persistently ELEVATED urine ALDOSTERONE
- 22. **Wielding scalpel**: PRIMARY HYPERALDOSTERONISM due to ALDOSTERONE-secreting ADENOMAS can be treated surgically (usually avoided in IDIOPATHIC HYPERALDOSTERONISM because BILATERAL)
- 23. **Discarded peanuts**: PRIMARY HYPERALDOSTERONISM can be treated with a low sodium diet (decreases blood volume)
- 24. **SPIRAL notebook**: PRIMARY & SECONDARY HYPERALDOSTERONISM can be treated with spironolactone (aldosterone ANTAGONIST)

Endocrine 2.3 - Adrenocortical Hyperfunction: Hyperaldosteronism & Hypercortisolism



- 25. **RAIN umbrella airship**: SECONDARY HYPERALDOSTERONISM (a result of increased RENIN → activates the renin-angiotensin-aldosterone system [RAAS])
- 26. Floppy heart airship: CHF can cause SECONDARY HYPERALDOSTERONISM (excessive peripheral edema → RAAS activation)
- 27. Liver crystalline rock : cirrhosis can cause SECONDARY HYPERALDOSTERONISM (excessive peripheral edema → RAAS activation)
- 28. Foaming from kidney container: nephrotic syndrome can cause SECONDARY HYPERALDOSTERONISM (excessive peripheral edema → RAAS activation)
- 29. Water falling into peripheral space : conditions with EXCESSIVE PERIPHERAL EDEMA (CHF, cirrhosis, nephrotic syndrome) can cause SECONDARY HYPERALDOSTERONISM (decreased intravascular volume \rightarrow decreased renal perfusion \rightarrow increased RENIN secretion by the kidney \rightarrow activation of RAAS \rightarrow elevated serum aldosterone)
- 30. **Creeping crab**: a RENIN-secreting TUMOR within the juxtaglomerular apparatus of the kidney can cause SECONDARY HYPERALDOSTERONISM (excessive RENIN → activation of RAAS)
- 31. **SPIRAL notebook**: PRIMARY & SECONDARY HYPERALDOSTERONISM can be treated with spironolactone (aldosterone ANTAGONIST)
- 32. Layers of fascicular mushroom trunks: zona FASCICULATA of the adrenal cortex (MIDDLE layer) (cells arranged in fascicles) (produces glucocorticoids)
- 33. **Over-sized moon-faced statue**: excessive glucocorticoids (cause Cushing's syndrome)
- 34. Large belly & skinny arms: excessive glucocorticoids cause central obesity and extremity wasting
- 35. Cracked statue: excessive glucocorticoids causes skin atrophy and striae
- 36. Elevated candy jar: excessive glucocorticoids cause glucose intolerance

- 37. "ADDition" light on adrenal cap: CORTISOL-secreting ADRENAL ADENOMA (most common cause of adrenal Cushing's syndrome)
- 38. **Light turned on**: CORTISOL-secreting ADRENAL ADENOMAS produces hormone (cortisol)
- 39. **Broken ACTHion clapperboard**: CORTISOL-secreting ADRENAL ADENOMAS lead to DECREASED ACTH (due to negative feedback on pituitary and hypothalamus) ("ACTH-independent")
- 40. **Shriveled yellow mushrooms**: CORTISOL-secreting ADRENAL ADENOMAS lead to ADRENAL ATROPHY (CONTRALATERAL side & non-tumor tissue on IPSILATERAL side) (due to lack of stimulation from ACTH) (specifically affects zona FASCICULATA)
- 41. Overgrowing mushroom nodules: NODULAR HYPERPLASIA of zona FASCICULATA (secretes CORTISOL) (causes adrenal Cushing's syndrome) (similar presentation as cortisol-secreting adrenal adenoma

Endocrine 2.4 - Cushing's Syndrome



- 1. Moon face : glucocorticoids
- 2. **Big CUSHION**: Cushing's syndrome (caused by excessive glucocorticoids
- Falling ACTHion clapper board: EXOGENOUS glucocorticoids lead to DECREASED adrenocorticotropic hormone (ACTH) (because of negative feedback on anterior pituitary)
- 4. **Shriveled adrenal rocks**: EXOGENOUS glucocorticoids cause adrenal gland atrophy (due to lack of stimulation from ACTH)
- Blown-up midsection: excessive glucocorticoids cause central obesity (hyperglycemia and insulin resistance → chronic insulin secretion → fat redistribution to trunk)
- 6. **Moon face reflection**: excessive glucocorticoids can cause "moon facies" (due to fat deposits in the face)
- 7. **Buffalo backpack**: excessive glucocorticoids can cause a "buffalo hump" (due to fat deposits in the neck and upper back)
- 8. Extracting candy moonstones from liver crater: excessive glucocorticoids can cause hyperglycemia (due to increased gluconeogenesis in the liver)
- 9. Broken key pad keeping astronaut from getting "INSIDE" : glucocorticoids can directly interfere with insulin signaling \rightarrow insulin resistance
- 10. Fat astronaut trying to get "INSIDE": glucocorticoids cause central obesity, which also contributes to insulin resistance
- 11. Oversized candy moonstone jar: excessive glucocorticoids can cause type 2 diabetes (due to hyperglycemia & insulin resistance)
- 12. **Helmet bursting steam** : excessive glucocorticoids can cause hypertension
- 13. **Large MINERAL crystals** : glucocorticoids have some intrinsic mineralocorticoid activity → increased sodium reabsorption and volume retention → hypertension
- 14. **Squeezing red tube**: glucocorticoids increase cardiovascular sensitivity to catecholamines (→ more significant VASOCONSTRICTION on exposure to epinephrine or norepinephrine)
- 15. **Blocking spiral bound notebook**: spironolactone (mineralocorticoid receptor antagonist) can be useful for the treatment of HTN in hypercortisolism (inhibit extra mineralocorticoid effects)
- 16. Spacesuit stretch marks : excessive glucocorticoids can cause purple striae (catabolism of collagen in subcutaneous tissues → stretch marks) (purple because of small capillary rupture)

- 17. **Straining to stand**: excessive glucocorticoids can cause central muscle weakness (e.g. difficulty lifting out of chair) due to catabolism of muscle
- 18. **Broken bone landing gear**: excessive glucocorticoids can cause osteoporosis (glucocorticoids increase bone resorption, decrease intestinal calcium absorption, decrease renal calcium reabsorption)
- 19. **Using crutches**: excessive glucocorticoids can cause an immunocompromised state (leads to increased infections and impaired wound healing)
- 20. **Torn suit**: excessive glucocorticoids can cause impaired wound healing (due to immunocompromised state)
- 21. Neutrophil first responders appearing from behind the hill: excessive glucocorticoids can cause increased neutrophil count (glucocorticoids lead to DEMARGINATION of neutrophils from endothelial surface)
- 22. Crying with cracked helmet: excessive glucocorticoids can present with psychiatric symptoms (depression, anxiety, irritability, labile mood)
- 23. Falling GardenR Hardware bag : excessive glucocorticoids INHIBIT GnRH release from the hypothalamus
- 24. **Irregular red oil drops**: excessive glucocorticoids can present with menstrual irregularities (decreased GnRH → decreased secretion of FSH and LH by pituitary → abnormal menstrual cycle)
- 25. **Pituitary pack containing ACTHion clapperboard**: Cushing's DISEASE (endogenous ACTH-dependent Cushing's syndrome due to an ACTH-secreting PITUITARY ADENOMA)
- 26. Raising ACTHion clapperboard : an ACTH-secreting pituitary adenoma causes ELEVATED ACTH levels → increased cortisol secretion from adrenal cortex
- 27. **Two oversized adrenal-esque rocks**: an ACTH-secreting PITUITARY ADENOMA causes BILATERAL adrenal gland hyperplasia (due to constant stimulation from ACTH)
- 28. Covered in pigmented moon mud: an ACTH-secreting PITUITARY ADENOMA can cause hyperpigmentation (due to effects of melanocyte stimulating hormone [MSH] cleaved from proopiomelanocortin [POMC])
- 29. **Moon mud on face and elbows**: in an ACTH-secreting PITUITARY ADENOMA, hyperpigmentation (due to overproduction of MSH) is most evident on sun exposed areas (face, neck) and trauma exposed areas (elbows, knees)
- 30. "Male" antenna: an ACTH-secreting PITUITARY ADENOMA can cause hyperandrogenism (ACTH stimulates zona reticularis → androgen production) which manifests in women as hirsutism, acne, and virilization

Endocrine 2.4 - Cushing's Syndrome



- 31. ADRENAL hat projecting MOON light: a CORTISOL-secreting ADRENAL TUMOR (adenoma or carcinoma) causes ENDOGENOUS Cushing's syndrome
- 32. Falling ACTHion clapper board: a CORTISOL-secreting ADRENAL TUMOR causes DECREASED ACTH (due to negative feedback on anterior pituitary) (ACTH-INDEPENDENT Cushing's syndrome)
- 33. Shriveled adrenal hats: a CORTISOL-secreting ADRENAL TUMOR causes adrenal gland atrophy (both contralateral side and remaining normal tissue on ipsilateral side) (specifically affects zona fasciculata)
- 34. Crab rover with ACTHion clapperboard : ECTOPIC ACTH-secreting TUMOR (an ENDOGENOUS Cushing's syndrome)
- 35. **Crab on chest badge**: ECTOPIC ACTH production is most commonly due lung tumors (especially small cell lung cancer and bronchial carcinoid tumors)
- 36. Raising ACTHion clapperboard ECTOPIC ACTH production causes ELEVATED ACTH levels → increased cortisol secretion by adrenal cortex :
- 37. **Moon mud**: ECTOPIC ACTH production causes hyperpigmentation (due to elevated MSH levels) (most evident on sun exposed and trauma exposed areas)
- 38. **Two oversized adrenal-esque rocks**: ECTOPIC ACTH production causes BILATERAL adrenal gland hyperplasia (due to constant stimulation from ACTH)
- 39. **Pee bag**: a 24-hour URINE free cortisol test can be used to diagnose HYPERcortisolism
- 40. **Salivating tongue**: late-night SALIVARY cortisol can be used to diagnose HYPERcortisolism (cortisol levels are not normally elevated in the evening)
- 41. **INDEX of Moonstones**: DEXAMethasone (a potent glucocorticoid) (administered as part of LOW and HIGH dose dexamethasone tests)
- 42. **Collecting SMALL moonstones near undisturbed pituitary pack**→ LOW dose dexamethasone inhibits physiologic hypercortisolism (but
 NOT pituitary or ectopic ACTH-secreting tumors) → negative feedback
 on hypothalamus and pituitary → decreased ACTH and cortisol:
- 43. Knocking down ACTHion from pituitary pack with LARGE moonstone: HIGH dose dexamethasone inhibits ACTH-secreting PITUITARY ADENOMAS (but NOT ectopic ACTH-secreting tumors) → negative feedback on hypothalamus and pituitary → decreased ACTH and cortisol

- 44. Losing LARGE moonstones to crab rover: HIGH dose dexamethasone is NOT sufficient to suppress ECTOPIC ACTH-producing tumors
- 45. **Moon flag scalpel**: ENDOGENOUS Cushing's SYNDROME is typically treated with SURGERY to remove tumor (with the exception of small cell carcinoma of the lung)
- 46. **Pinecone KEY**: HYPERcortisolism can be treated with ketoconazole (inhibits adrenal hormone production)
- 47. WhiFFle ball: HYPERcortisolism can be treated with mifepristone (antagonizes cortisol and progesterone receptors)
- 48. **Make-up**: HYPERcortisolism can be treated with metyrapone (inhibits 11-hydroxylase)
- 49. **MITosis pendant**: HYPERcortisolism can be treated with mitotane (directly suppresses adrenal glands)
- 50. **Moon flag scalpel**: HYPERcortisolism can be treated with bilateral adrenalectomy (requires lifelong glucocorticoid and mineralocorticoid replacement)
- 51. **Nelson Armstrong with pituitary bag & ACTHion clapperboard**: Nelson syndrome (sudden drop in cortisol following bilateral adrenalectomy → increased ACTH secretion → ENLARGEMENT of PITUITARY ADENOMA)
- 52. Clutching head & seeing stars: Nelson syndrome can present with headache and visual disturbances (enlargement of PITUITARY ADENOMA → increased pressure on sella turcica)
- 53. **Pigmented moon mud**: Nelson syndrome can present with hyperpigmentation (increased ACTH secretion is secondary to increased POMC synthesis → also results in increased production of MSH)

Endocrine 2.5 - Pheochromocytoma & Neuroblastoma



- 1. "CENTRAL PARK": adrenal medulla (the center of the adrenal gland)
- 2. CREST : the adrenal medulla is composed of chromaffin cells, which derive from the NEURAL CREST
- 3. **Nerve wires**: the adrenal medulla is innervated by preganglionic sympathetic fibers (secretes catecholamines when stimulated)
- "FROZEN COLORS": PHEOCHROMOCYTOMAS (the most common medullary adrenal tumor in adults) (composed of neoplastic chromaffin cells)
- 5. "10 for \$10" crab with 2 shaved ice servings: 10% of pheochromocytomas are malignant, 10% are bilateral, 10% occur outside the adrenal gland
- Frozen color bags along SIDES OF CENTER POST:
 PARAGANGLIOMAS (pheochromocytomas outside adrenal gland) (occur near sympathetic chain ganglia [on either side of SPINAL COLUMN])
- 7. **Two adrenal umbrellas**: FAMILIAL pheochromocytomas are more likely to be bilateral
- 8. "2 MEN": pheochromocytomas are common in MEN2 (syndrome characterized by multiple endocrine tumors) (caused by mutations in the RET protooncogene)
- 9. "A family tradition" domino : MEN2 and VHL syndromes are autosomal dominant
- 10. "VHL" motor company: Von Hippel Lindau (VHL) syndrome is associated with pheochromocytomas and paragangliomas (caused by mutations in VHL tumor suppressor gene)
- 11. Curly red hair : VHL syndrome is associated with hemangioblastomas $\,$
- 12. "RENASCAR" jacket with side crab pockets: VHL syndrome is associated with clear cell RENAL cell carcinoma
- 13. "1 nuff" sign: NEUROFIBROMATOSIS 1 (NF-1) is associated with pheochromocytomas (usually unilateral) (caused by mutations in the NF1 tumor suppressor gene)
- 14. "Cafe au lait" cart: NF-1 is associated with cafe au lait spots
- 15. Foam bits: NF-1 is associated with neurofibromas
- ${\bf 16. \ North \ compass}: pheochromocytomas \ secrete \ norepine phrine$
- 17. "EPIC FLAVOR": pheochromocytomas secrete epinephrine
- 18. Dopamine rope : pheochromocytomas secrete dopamine
- 19. **Brain freeze**: pheochromocytomas can present with headaches
- 20. Impatiently holding heart watch: pheochromocytomas can present with tachycardia
- 21. **Looking nervous**: pheochromocytomas can present with sweating
- 22. **Steaming angry**: pheochromocytomas can present with hypertension
- 23. **Relieved after brain freeze**: in pheochromocytomas, symptoms are episodic

- 24. Yellow shaved ice: pheochromocytomas appear yellow-tan with well-defined margins
- 25. Cherry sauce with skull shirt: pheochromocytomas eventually develop areas of hemorrhage, necrosis, and cyst formation
- 26. Piles of spindle-y cells: pheochromocytomas display nests of spindle-like chromaffin cells ("Zellballen")
- 27. **Sprinkles**: pheochromocytoma cells display a granular cytoplasm (due to the presence of catecholamine granules)
- 28. **Urinary and sanguineus syrups**: pheochromocytomas can be diagnosed by measuring urinary or plasma metanephrines or catecholamines
- 29. **METAL toppings**: METAnephrines (metanephrine, normetanephrine, vanillylmandelic acid) are metabolites of catecholamines
- 30. Scalpel pillars: pheochromocytomas are treated with surgical removal
- 31. **PHOENIX talons grasping ALPHA**: pheochromocytomas are treated with PHENoxybenzamine (a non-selective ALPHA blocker)
- 32. **Mute blocking BETA bugle**: pheochromocytomas are treated with BETA blockers only AFTER phenoxybenzamine has been administered
- 33. **Bugle player with steaming ears**: treatment of pheochromocytomas with a beta blocker ALONE leads to severe hypertension (due to unopposed alpha-1 activity)
- 34. "Neuro-blaster" children's toy: NEUROBLASTOMA (the most common adrenal medulla tumor in children)
- 35. **CREST**: neuroblastomas arise from neural crest cells (populate the adrenal medulla and sympathetic ganglia)
- 36. **Blast toys lining SIDES of CENTER POST**: neuroblastomas can develop anywhere along the sympathetic chain ganglia (although most common in adrenal medulla)
- 37. **n-MICE**: some neuroblastomas contain amplification of the oncogene N-MYC (a DNA transcription factor)
- 38. Clutching abdomen: neuroblastomas can present with abdominal pain
- 39. **Pockets stuffed with neuro-blasters**: neuroblastomas can present with an abdominal mass
- 40. Flame bandana: neuroblastomas can present with fever
- 41. Skinny kid: neuroblastomas can present with weight loss
- 42. **Bone injured by blast**: neuroblastomas may present with bone pain (due to bone metastasis)
- 43. **Uncontrollable dancing and crazy eyes**: neuroblastomas can present with opsoclonus-myoclonus syndrome (disorganized eye movement, myoclonus (jerking of extremities), ataxia
- 44. Wheel of small blue firecrackers: neuroblastomas display small round blue cells organized around a central space (creates "Homer-Wright" rosettes)

Endocrine 3.1 - Diabetes Mellitus: Pathogenesis & Acute Clinical Manifestations



- Racecar #1 with cracked BETAscotch wheels: type 1 diabetes mellitus (T1DM) (results from dysfunction of pancreatic BETA cells)
- 2. Kid driver: T1DM typically presents in childhood
- Antibody wrench embedded in cracked BETAscotch wheel: T1DM is caused by autoimmune destruction of BETA cells (in the pancreatic islets of Langerhans)
- Rolled-up "INSULIN" mat: T1DM is characterized by absolute INSULIN DEFICIENCY (due to destruction of pancreatic beta cells)
- 5. "3-Crusader's" bar: in T1DM, CD8+ killer T-cells attack pancreatic beta cells (occurs as a result of T cells losing "self-tolerance")
- 6. Antibody archers: in T1DM, B-cells produce autoantibodies against pancreatic beta cells (as a result of activation by CD4+ helper T cells)
- 7. "Glutamates" on archer candy box : anti-GLUTAMIC ACID decarboxylase antibodies are common in T1DM
- 8. "Inside" on archer candy box : anti-INSULIN antibodies are common in T1DM
- 9. "1234" start lights: T1DM is associated with polymorphisms in MHC class 2 molecules (particularly haplotypes HLA-DR3 and DR4 [which are prone to presenting self-antigens to helper T cells])
- Additional antibody wrenches: T1DM is associated with other autoimmune diseases (Grave's disease, Hashimoto's thyroiditis, celiac disease)
- 11. Pale ISLANDS surrounded by darker candy box: the ISLETS of Langerhans (which contain endocrine cells) appear pale compared to the acinar cells (which have exocrine function) (NORMAL pancreatic histology)
- 12. "BLUE DOTS": in T1DM, pancreatic tissue displays a lymphocytic infiltrate (appears as dense collection of BLUE cells) (composed primarily of T-cells)
- 13. **Squashed ISLAND candies**: T1DM leads to a decreased number and size of ISLETS (due to the autoimmune process)
- 14. Candy bouncing off purple tray: in T1DM, glucose is unable to enter peripheral cells (because of insulin deficiency)
- 15. **Munching on cotton candy in striated cup**: in T1DM, GLYCOGENOLYSIS in the liver and skeletal muscle INCREASES (as part of catabolic state from insulin deficiency) → increased serum glucose
- 16. Raising candy jar: T1DM leads to HYPERGLYCEMIA (due to insulin deficiency)

- 17. **Pulling candy from liver jar** : in T1DM, GLUCONEOGENESIS in the liver INCREASES (as part of catabolic state from insulin deficiency) \rightarrow increased serum glucose
- 18. **Spilling candy-laden drink**: T1DM presents with GLUCOSURIA (severe hyperglycemia exceeds renal reabsorption capacity for glucose)
- 19. Large amount of yellow drink : T1DM presents with POLYURIA (loss of glucose in urine \rightarrow excessive loss of water in urine)
- 20. **High condOSMents station**: T1DM presents with INCREASED PLASMA OSMOLALITY (severe hyperglycemia → polyuria + increased concentration of solutes in plasma)
- 21. **Scarfing down liquids**: T1DM presents with POLYDIPSIA (excessive thirst) (increased plasma hyperosmolality triggers osmoreceptors in the hypothalamus)
- 22. **Breaking down protein bar**: T1DM leads to breakdown of muscle into amino acids → travel to liver to supply gluconeogenesis) (due to insulin deficiency)
- 23. **Breaking down fatty donuts**: T1DM leads to increased LIPOLYSIS (insulin deficiency → decreased inhibition of hormone sensitive lipase)
- 24. **Thin spectator**: T1DM presents with weight loss (due to fat and muscle breakdown & inability of peripheral tissues to absorb glucose)
- 25. **Face stuffed**: T1DM presents with excessive hunger despite weight loss (due to inability of peripheral tissues to absorb glucose)
- 26. Candy KEY: T1DM can cause diabetic KETOacidosis (a severe hyperglycemia crisis) (characterized by formation of ketone bodies)
- 27. Bringing in NEW "INSULIN" mat: T1DM is treated with exogenous including
- 28. Racecar #2: type 2 diabetes mellitus (T2DM) (characterized by RELATIVE insulin deficiency [due to combination of insulin resistance & beta cell dysfunction])
- 29. **RESISTING pull on "INSIDE" mat**: in T2DM, INSULIN RESISTANCE contributes to the RELATIVE insulin deficiency
- 30. **Broken BETAscotch candies**: in T2DM, pancreatic BETA CELL DYSFUNCTION contributes to the RELATIVE insulin deficiency
- 31. **Family of spectators** : family history is an important risk factor for T2DM
- 32. **Oversized belly stuck in car**: obesity is an important risk factor for INSULIN RESISTANCE (particularly ABDOMINAL OBESITY)
- 33. Releasing cytoCOINS from fatty wall : adipocytes release CYTOKINES ("adipokines") → imbalance contributes to insulin resistance [contributes to INSULIN RESISTANCE in OBESITY]

Endocrine 3.1 - Diabetes Mellitus: Pathogenesis & Acute Clinical Manifestations



- 34. **Breaking SIGNAL flag at fatty acid fence**: intracellular triglycerides and free fatty acids inhibit insulin SIGNALING [contributes to INSULIN RESISTANCE in OBESITY]
- 35. CYTOcoins at fatty acid fence: free fatty acids within macrophages and beta cells leads to CYTOKINE release → insulin resistance [contributes to INSULIN RESISTANCE in OBESITY]
- 36. Increasing "INSIDE" mats: in T2DM, pancreatic beta cells initially INCREASE INSULIN PRODUCTION in response to insulin resistance
- 37. Collapsing over excessive stack of "INSIDE" mats: in T2DM, pancreatic beta cells are eventually unable to sustain a state of increased insulin production ("BETA CELL BURNOUT") → relative insulin deficiency
- 38. Candy debris and fatty acid tread on BETAscotch wheel :
 excessive glucose and free fatty acids accumulate in the beta cells →
 CYTOKINE release → contributes to BETA CELL DYSFUNCTION
- "GLP!": in T2DM, GLP (an incretin secreted by the intestines) may inadequately stimulate beta cells (may contribute to BETA CELL DYSFUNCTION)
- 40. "AMY LIN" riding adjacent to "INSIDE" mat: AMYLIN (a polypeptide that is co-secreted with insulin from beta cells) (normally assists in glycemic control)
- 41. **Pink gunk mucking up BETAscotch wheels**: in T2DM, AMYLIN forms aggregates of AMYLOID inside beta cell secretory granules → eventually deposits throughout islets (may contribute to beta cell "burnout" OR may be a consequence of "burn out")
- 42. **BETAscotch wheel leaving the scene**: T2DM is associated with LOSS of beta cells (although less important than beta cell dysfunction)
- 43. **Gingerbread grandfather clock**: T2DM may present with chronic symptoms (e.g. micro/macrovascular disease, neuropathy)
- 44. "**HHONK**": Hyperosmolar Hyperglycemic State (HHS) (alternatively "HHONK") is a complication of T2DM characterized by marked HYPERglycemia and HYPERosmolality
- 45. **Healthy runner**: T2DM is treated with lifestyle modifications (weight loss, physical activity, dietary changes)
- 46. Consuming pills: T2DM is often treated with oral medications
- 47. "Candy 200" with 2 RANDOMLY checkered flags: DM can be diagnosed with a random serum glucose level of ≥ 200 mg/dL on 2 separate occasions (1 occasion if symptomatic)
- 48. **2 "FASTEST lap signs** : 1:26": DM can be diagnosed with a FASTING serum glucose level of ≥ 126 mg/dL on 2 separate occasions

- 49. Consuming candy before "2 HOUR race to the cup!": DM can be diagnosed with a 2 hour glucose tolerance test (75 mg glucose administered orally) (DM diagnosed if serum glucose after 2 hours is ≥ 200 mg/dL)
- 50. "A1" car with hubcaps and streamers: HEMOGLOBIN A1c [HbA1c] (an alternative term for GLYCATED hemoglobin) reflects glucose control over the past 3 months (because of 3-month RBC lifespan)
- 51. **"6" and "5" car design** : DM can be diagnosed with a HbA1c ≥ 6.5%
- 52. **Metabolic brownies**: METABOLIC SYNDROME (a constellation of signs and symptoms including obesity, hyperglycemia, hypertension, and dyslipidemia)
- 53. "X" candy spears : Syndrome X (an alternative term for METABOLIC SYNDROME)
- 54. **Oversized abdomen**: abdominal obesity is a feature of METABOLIC SYNDROME
- 55. Candy jar on raised balcony: hyperglycemia is a feature of METABOLIC SYNDROME56. Steaming angry: hypertension is a feature of METABOLIC
- SYNDROME
- 57. Large butter slab: dyslipidemia (high triglycerides or low HDL) is a feature of METABOLIC SYNDROME
- 58. **Family gathering**: a family history is a risk factor for METABOLIC SYNDROME
- 59. **Measuring expanded abdomen**: in METABOLIC SYNDROME (as well as DM), excessive visceral abdominal adipose tissue (measured as waist circumference) contributes to INSULIN RESISTANCE
- 60. **Raising 3-pronged fork**: a diagnosis of METABOLIC SYNDROME requires at least 3 of 5 findings (elevated fasting glucose, hypertension, hypertriglyceridemia, hypercholesterolemia, elevated waist circumference)
- 61. Coronary crown & broken heart lute: METABOLIC SYNDROME is a significant risk factor for cardiovascular disease (2x more likely)
- 62. **Metabolic king pointing to racecar #2**: METABOLIC SYNDROME is a significant risk factor for T2DM (3-5x more likely)
- 63. **MOODY prince**: Mature-Onset Diabetes of Youth (MODY) (a genetic syndrome characterized by non-insulin dependent DM at a young age)
- 64. **Pointing to racecar #2**: MODY is most similar to T2DM (INSULIN RESISTANCE is prominent in both)
- 65. Domino: MODY is autosomal dominant

Endocrine 3.2 - Diabetic Ketoacidosis (DKA) & Hyperosmolar Hyperglycemic State (HHS)





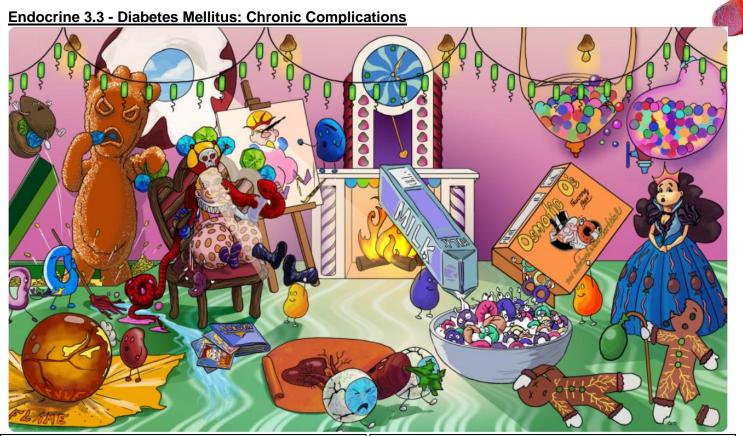
- 1. #1 car with broken BETAscotch candies: type I diabetes (T1DM) (a result of autoimmune destruction of pancreatic BETA cells)
- 2. Grand prize KEY: diabetic KETOacidosis (DKA) (occurs in T1DM)
- 3. Torn "INSIDE" mat: T1DM is characterized by absolute INSULIN DEFICIENCY
- 4. **Bacterial lanterns**: DKA can be precipitated by acute stressors (such as infection [oneumonia, UTI, sepsis])
- 5. "Candy 200": in DKA, serum glucose is > 200 mg/dL (often 350-500) (rapid onset)
- 6. Raised "Glucagon" sweetener packet: INSULIN DEFICIENCY leads to GLUCAGON EXCESS (due to loss of normal inhibition of glucagon secretion by insulin) (promotes a CATABOLIC state)
- 7. Raised "EPIC" sign: STRESS causes INCREASED secretion of EPINEPHRINE (contributes to CATABOLIC state [particularly in the absence of insulin])
- 8. Moon faced marshmallows: STRESS causes INCREASED secretion of CORTISOL (contributes to CATABOLIC state [particularly in the absence of insulin])
- 9. **Breaking down cotton candy in striated cup**: GLUCAGON stimulates GLYCOGENOLYSIS (glycogen breakdown) in the liver and skeletal muscle increases serum glucose (a short-lived effect)
- 10. Candy escaping liver jar : GLUCAGON stimulates GLUCONEOGENESIS in liver and kidneys \rightarrow increases serum glucose
- 11. Overflowing yellow exhaust with candy debris : severe HYPERGLYCEMIA leads to OSMOTIC DIURESIS (serum glucose > 200 mg/dL \rightarrow exceeds renal glucose reabsorption capacity \rightarrow GLUCOSURIA \rightarrow POLYURIA)
- 12. Fainting next to empty tank : OSMOTIC DIURESIS in DKA can lead to signs of volume depletion (e.g. hypotension)
- 13. Cracked empty kidney tank : OSMOTIC DIURESIS in DKA can lead to acute kidney injury (AKI) (volume depletion \rightarrow decreased renal perfusion \rightarrow prerenal azotemia)
- 14. **Depleted peanut shell in sugary water** : HYPERGLYCEMIA (e.g. DKA or HHS) can cause HYPONATREMIA (elevated serum glucose \rightarrow osmotic shift of water from intracellular to extracellular compartments \rightarrow dilution of serum sodium)
- 15. Breaking down fatty donuts : INSULIN DEFICIENCY leads to LIPOLYSIS (breakdown of lipids) (lack of insulin \to no inhibition of hormone-sensitive lipase)
- 16. **Raised TRIDENT**: DKA can present with hypercholesterolemia and triglyceridemia (insulin deficiency \rightarrow lipolysis \rightarrow lipids released into bloodstream)
- 17. Fatty acid fence : in DKA, breakdown of lipids leads to increased FREE FATTY ACIDS
- 18. **BETA candies in LIVER packaging** : free fatty acids undergo BETA OXIDATION in liver mitochondria
- 19. **Elevated rAce CoAch**: ACETYL CoA is produced from free fatty acids (via beta oxidation)
- 20. **Key to the candy jar**: in DKA, KETONE BODIES accumulate in the serum (produced from ACETYL CoA via KETOGENESIS) (used as fuel in peripheral tissues because glucose is unable to enter cells without insulin)
- 21. "Sweet 'n ACID" SOUR candies: DKA leads to accumulation of ACETOACETATE (a ketone body [specifically a ketoACID])
- 22. **Sweet One**": DKA leads to accumulation of ACETONE (a ketone body [a true ketone])

- 23. Fruity aroma: ACETONE has a sweet odor
- 24. "Big cHew Bubble" SOUR gum: BETA-HYDROXYBUTYRATE (a ketone body [specifically a hydroxy ACID])
- 25. **Sour candies in yellow puddle**: in DKA, KETONE bodies are excreted in urine (urine screen for beta-hydroxybutyrate is initial test for DKA)
- 26. **Spewing lemonade volcano**: DKA causes a METABOLIC ACIDOSIS (a result of CO2 produced by ketogenesis & KETOACIDS) (INCREASED ANION GAP due to ketoacids)
- 27. **Deep breath of cookie smell**: DKA presents with KUSSMAUL breathing (rapid, deep breaths to blow off CO2) (to compensate for METABOLIC ACIDOSIS)
- 28. Raising BANANA phrosting out of container: in hyperglycemia, potassium moves from the intracellular to extracellular space due to fluid shifts (DKA/HHS), H+buffer system (DKA), or decreased insulin activity (DKA/HHS) → normal or high serum potassium
- 29. **Depleted BANANA phrosting container**: hyperglycemia (e.g. DKA or HHS) causes LOW TOTAL BODY POTASSIUM (depleted intracellular potassium) despite normal or high serum potassium
- 30. **Depleted banana PHROSTING container**: hyperglycemia (e.g. DKA or HHS) causes LOW TOTAL BODY PHOSPHATE (depleted intracellular phosphate) despite normal or high serum phosphate
- 31. Nauseated: DKA can present with nausea, vomiting, and abdominal pain
- 32. Fluid repletion: IV fluids are part of the initial management of DKA (in order to correct hypovolemia)
- 33. **Administering bananas**: potassium replacement is part of the initial management of DKA (in order to correct low total body potassium)
- 34. "INSIDE" mat: IV insulin administration is part of management of DKA
- 35. "HHONK": Hyperosmolar Hyperglycemic NonKetotic State (alternatively Hyperosmolar Hyperglycemic State [HHS]) is characterized by marked HYPERglycemia and HYPERosmolality
- 36. **#2 car with intact BETAscotch candies**: HHS primarily occurs in type 2 diabetics (T2DM)
- 37. Elderly woman unable to access water: HHS is most common in older, debilitated patients who are unable to access sufficient water (HHS has SLOW onset; a mobile person may have time to correct the developing hyperglycemia by increasing hydration)
- 38. Oversized "1 GRAND" bar: HHS is associated with profound hyperglycemia (glucose is often > 1,000 mg/dL)
- 39. Completely empty water tank : in HHS, severe HYPERGLYCEMIA leads to volume depletion (due to OSMOTIC DIURESIS) (same mechanism as DKA)
- 40. **Lightning bolt**: HHS can present with hypotension and hypovolemic shock (due to severe volume depletion)
- 41. Excessive red condOSMments : in HHS, extreme HYPERGLYCEMIA leads to severe HYPEROSMOLALITY (320-380 mOsm)
- 42. Cracked helmet : HHS can present with neurologic symptoms (seizures, coma) (severe hyperosmolality \rightarrow fluid shifts in the brain)
- 43. Failed to reach sour candy reward : HHS does NOT cause ketoacidosis (type 2 diabetics produce some insulin \rightarrow inhibits hormone sensitive lipase \rightarrow no lipolysis or fatty acid breakdown \rightarrow no ketone bodies)



- 1. **Hexose candy binding**: glucose can form covalent bonds with free amino groups on proteins WITHOUT the need for an enzyme (non-enzymatic glycation)→ advanced glycation end products (AGE) (degree of AGE formation is DIRECTLY correlated with glucose levels)
- Jerky links: AGEs can crosslink proteins, causing damage or dysfunction (especially in collagen) (in arteries, it can reduce intimal integrity, allowing LDL to enter vessel walls and speed atherosclerosis)
- 3. Raging gummy bear eating candy: AGEs can bind to and active receptors of AGE (RAGE) throughout the body
- 4. $\textbf{Cytocoins}: \mathsf{RAGE}$ activation can lead to release of growth factors and inflammatory cytokines
- 5. **Damaging sugar crystals**: RAGE mediated inflammation can lead to the generation of reactive oxygen species→ diffuse tissue damage (particularly to vascular endothelial cells)
- 6. **Candy inside**: AGE WITHIN endothelial cells cause release of inflammatory mediators, reactive oxygen species, and growth factors (growth factors promote the local production of excess collagen)
- 7. **Thick basement door**: on histology, diabetic MICROangiopathy is associated with diffuse basement membrane thickening in capillaries and small arterioles
- 8. **4 candy sharks**: the diffuse basement membrane thickening in diabetic MICROangiopathy is caused by overproduction of type IV collagen by vascular fibroblasts
- 9. **Pink gummy ring**: on histology, diabetic MICROangiopathy is associated with concentric layers of eosinophilic hyaline around the basement membranes of capillaries
- 10. **Mopping leak**: diffuse basement membrane thickening reduces the integrity of capillaries → leakage of plasma proteins (diabetic MICROangiopathy)
- 11. **Shriveled kidney raisin**: diabetic nephropathy is a leading cause of chronic kidney disease
- 12. **Glomerular tail near think basement door**: diffuse glomerular basement membrane thickening (a feature of diabetic NEPHROPATHY)
- 13. **Glomerular tail with pink goo**: glomerulosclerosis (hyaline deposition in leaky glomerular capillaries) (a feature of diabetic NEPHROPATHY)
- 14. **Wet album**: leaky capillaries in diabetic nephropathy spill albumin into urine (microscopic albuminuria is an early marker of diabetic kidney

- disease) (late renal disease may manifest as severe albuminuria with reduced GFR)
- 15. **Hollow jelly bean eyes**: diabetic RETINOPATHY is one of the leading causes of blindness in the United States (proliferative [new vessel formation] and nonproliferative [no new vessels])
- 16. **Red cinnamon flecks**: microaneurysms (early sign of nonproliferative diabetic RETINOPATHY)
- 17. **Flame cinnamon candy**: retinal blood vessel rupture in diabetic retinopathy leads to flame hemorrhages (nonproliferative diabetic RETINOPATHY)
- 18. **Cotton cloth**: "cotton wool spots" (fluffy-white appearing areas caused by micro-infarctions of the nerve fiber layer) (nonproliferative diabetic RETINOPATHY)
- 19. **Vascular VEGGIE roots**: PROLIFERATIVE diabetic retinopathy involves neovascularization of the retina due to increase in VEGF
- 20. **Nodular pink candies** : hyaline deposition in leaky glomerular capillaries \rightarrow Kimmelstiel-Wilson nodules (a feature of NODULAR glomerulosclerosis)
- 21. **Veggie juice on retinal rug**: neovascularization in PROLIFERATIVE diabetic retinopathy is disorganized and prone to hemorrhage (blood can enter vitreous substance \rightarrow cloudy vision)
- 22. Rock-hard eyeball candy : neovascularization in PROLIFERATIVE diabetic retinopathy can block drainage of aqueous humor \to glaucoma
- 23. Rolled up rug: PROLIFERATIVE diabetic retinopathy can lead to retinal detachment
- 24. **Sclerotic gummy snake**: hyaline arteriolosclerosis (deposition of hyaline material in arterioles causing wall thickening and luminal narrowing)
- 25. Atheromatous frosting in arterial donut: diabetes is a risk factor for atherosclerosis (chronic inflammation and lipid oxidation + AGE disruption of endothelium → plaque formation)
- 26. Coronary crown with yellow dome : diabetes increases the risk of coronary artery disease (CAD) \to MI, and heart failure
- 27. **Skull in crown**: cardiovascular disease is the most common cause of death in diabetes
- 28. **Black stroke on head** : diabetes increases the risk of atherosclerosis → cerebrovascular disease → stroke



- 29. "Sweetened with Sorbitol": many cells trap glucose by converting it to sorbitol (via aldose reductase)
- 30. "Fructose free": some tissues (retina, lens, Schwann cells) lack sorbitol dehydrogenase and are unable to convert sorbitol to fructose
- 31. **Milk on osmotic Os**: sorbitol is osmotically active and causes cellular edema in tissues that lack sorbitol dehydrogenase (retina, lens, Schwann cells)
- 32. **Blurry monocle**: sorbitol accumulation causes osmotic damage to the lens→ protein precipitation→ cataracts
- 33. **Stocking and glove frosting**: osmotic damage to Schwann cells causes demyelination→ "stocking glove" peripheral neuropathy (loss of proprioception and vibration [large nerves] followed by loss of pain and temperature [small fibers]
- 34. **Crumbled heel**: unrecognized trauma (due to peripheral neuropathy), peripheral arterial disease, and immune dysfunction→ diabetic foot ULCERS
- 35. Removing black boot : secondary infection of diabetic foot ulcers—gangrene (may require amputation)
- 36. Crushed chocolate joint : unperceived joint damage due to peripheral neuropathy in diabetes \to Charcot joint
- 37. Cranial bite and broken eye: diabetic neuropathy can cause cranial nerve palsies (especially affecting extraocular muscles [CN III, IV, and VI])
- 38. Candy stomach stop valve: diabetes can damage the vagus nerve and cause gastroparesis→ early satiety, abdominal pain, nausea, bloating
- 39. Floppy lollipop: autonomic neuropathy in diabetes can lead to impotence
- 40. **Bladder candy strop valve**: autonomic neuropathy in diabetes can lead to neurogenic bladder→ incomplete voiding→ overflow incontinence
- 41. Fainting : autonomic neuropathy in diabetes can lead to orthostatic hypotension— syncope

- 42. **Black velvety ruffles**: acanthosis nigricans (velvety pigmented skin plaques, especially in the neck, back, and axillae seen in chronic diabetes)
- 43. **Ulcerated candies**: necrobiosis lipoidica (small yellow-brown papules that progress to erosions and ulcerations seen in chronic diabetes)
- 44. **Bacterial and fungal lanterns**: diabetics are prone to recurrent infection (due to poor wound healing, urinary retention, neutrophil dysfunction, and high blood glucose)
- 45. **Fungal lantern**: diabetics are prone to recurrent fungal infections (e.g. mucocutaneous candidiasis, and rhinocerebral mucormycosis) due to high blood glucose
- 46. **Bacterial lantern**: diabetics are prone to recurrent bacterial infections (e.g. malignant otitis externa due to Pseudomonas infection)

Endocrine 4.1 - Hypothyroidism: Overview & Hashimoto's Thyroiditis



- 1. UNTIED bowtie: HYPOthyroidism
- 2. Fallen "BMR" bar : low thyroid hormone leads to low basal metabolic rate (BMR)
- 3. **Spilled ice** : hypothyroidism can present with cold intolerance (decreased BMR ightarrow decreased heat production)
- 4. "COOL DRY" place : hypothyroidism can present with cool dry skin
- 5. Messy hair: hypothyroidism can present with brittle hair
- 6. **Puffy eyes**: hypothyroidism can present with periorbital edema (due to myxedema)
- 7. GAG: glycosaminoglycans (such as hyaluronic acid)
- 8. **Drink MIXer**: MYXedema (non-pitting edema caused by accumulation of glycosaminoglycans)
- 9. **Sticking out tongue**: hypothyroidism can present with macroglossia (tongue enlargement) (due to myxedema)
- 10. **Compressed wrist**: hypothyroidism can present with hand numbness, tingling, or pain (first 3 fingers and half of 4th finger) (myxedema in carpal tunnel → median nerve compression)
- 11. Fat belly: hypothyroidism can present with weight gain (due to decreased BMR and non-pitting edema)
- 12. Low-density gravy boat on broken saucer: decreased thyroid hormone is associated with downregulation of LDL receptors in peripheral tissues
- 13. Full stick of butter : hypothyroidism can present with hypercholesterolemia (downregulation of LDL receptors \rightarrow accumulation of LDL in the blood
- 14. **Plunger**: hypothyroidism can present with constipation (due to decreased GI motility)
- 15. **Slumped over**: hypothyroidism can present with hypothyroid myopathy (proximal muscle weakness, cramps, myalgias)
- 16. **Torn red stripes**: in hypothyroid myopathy, fast twitch type II fibers undergo atrophy (fast twitch rely on glycolysis for energy → therefore particularly affected by decreased BMR)
- 17. Raised crisPy chiCKen: hypothyroid myopathy presents with increased serum creatinine kinase (CK)
- 18. **Broken "Bugle Boy" whiskey**: decreased thyroid hormone leads to downregulation of BETA adrenergic receptors in the heart

- 19. Fallen heart watch: hypothyroidism can present with bradycardia, decreased cardiac contractility, and decreased cardiac output (due to down-regulation of beta receptors & impaired calcium flux in cardiac myocytes)
- 20. **Difficulty breathing**: hypothyroidism can present with dyspnea on exertion (due to decreased cardiac output)
- 21. **Tight peripheral bands**: hypothyroidism can increase systemic vascular resistance (SVR) (decreased thyroid hormone → impaired relaxation of vascular smooth muscle)
- 22. **Steaming angry**: hypothyroidism can present with hypertension (due to increased SVR)
- 23. "Coronary crown" whiskey: hypothyroidism can contribute to development of atherosclerosis (due to dyslipidemia, endothelial dysfunction)
- 24. **Floppy heart balloon**: hypothyroidism can contribute to development of heart failure (due to hypertension and coronary artery disease)
- 25. Irregular red juice: hypothyroidism can present with irregular menses
- 26. **Kid with falling bowtie**: congenital hypothyroidism (cretinism) (caused by insufficient thyroid hormone in-utero) (due to fetal thyroid dysgenesis, maternal hypothyroidism, or iodine deficiency) presents with intellectual disability
- 27. **Bulging belly**: cretinism can present with pot-belly and protruding umbilicus
- 28. Pale puffed cheeks: cretinism can present with pallor and puffy face
- 29. Sticking out tongue: cretinism can present with protuberant tongue
- 30. **Stockings & gloves**: hypothyroidism can presents with distal symmetric neuropathy (sensory loss, tingling, pain) in "stocking glove" distribution
- 31. Slowly falling triangular mixer: hypothyroidism can present with LOSS or DELAY of deep tendon reflexes
- 32. **Tearful bartender**: hypothyroidism can present with depression, anxiety, or cognitive impairment

Endocrine 4.1 - Hypothyroidism: Overview & Hashimoto's Thyroiditis



- 33. **Comatose mixologist**: myxedema coma (exaggerated form of hypothyroidism) (due to longstanding severe hypothyroidism OR an acute stressor)
- 34. **Cold & confused** : myxedema coma presents with confusion, lethargy, and hypothermia (may progress to coma)
- 35. **Collapsed** : myxedema coma can lead to cardiovascular collapse (severe hypothyroidism \rightarrow hypotension and decreased cardiac output)
- 36. Falling peanuts: myxedema coma may present with HYPOnatremia (due to severe hypothyroidism)
- 37. **Spilled candy jar**: myxedema coma can present with HYPOglycemia (due to severe hypothyroidism or concomitant adrenal insufficiency)
- 38. **Torn adrenal beanie**: hypothyroidism may be associated with adrenal insufficiency (due to corresponding autoimmune disease or dysfunctional pituitary/hypothalamus)
- 39. Falling T4 time bomb: in PRIMARY hypothyroidism, T4 is LOW (in addition to ELEVATED TSH)
- 40. Tucking away T4 time bomb : in SUBclinical hypothyroidism, TSH is ELEVATED but T4 is NORMAL
- 41. **Torn HYPE poster & knocked over #3**: TERTIARY (CENTRAL) hypothyroidism is caused by hypothalamic dysfunction
- 42. **Bashed pituitary punching bag & knocked over #2**: SECONDARY (CENTRAL) hypothyroidism is caused by pituitary dysfunction
- 43. Falling T4 time bomb : in CENTRAL hypothyroidism, T4 is LOW (due to decreased secretion of TSH by pituitary)
- 44. **Tightening bowtie while crouching LOW**: in CENTRAL hypothyroidism, TSH is LOW (due to decreased secretion from pituitary)
- 45. "T4" plastic explosive: hypothyroidism is treated with oral levothyroxine (synthetic T4)
- 46. **Dr. Hashimoto**: Hashimoto's thyroiditis (chronic lymphocytic thyroiditis) (most common cause of hypothyroidism in iodine-sufficient parts of the world)
- 47. **Antibody hands & undone neck tie**: Hashimoto's thyroiditis is caused by autoimmune destruction of the thyroid gland \rightarrow irreversible hypothyroidism
- 48. **Older female agent**: Hashimoto's thyroiditis is most common in older women (45-65 years of age)
- 49. **Array of antibody tools**: Hashimoto's thyroiditis is associated with other autoimmune disorders (Celiac disease, Type 1 DM, primary adrenal insufficiency)

- 50. **HospitAList DR Ha5h3moto**: Hashimoto's thyroiditis is associated with HLA-DR3 and DR5 (MHC class 2 genes)
- 51. Killer T-cell crusader : CD8+ cytotoxic T cells directly damage thyroid follicular cells → contribute to development of Hashimoto's thyroiditis
- 52. "Squire" Vodka surrounded by cytoCOINS and macroCAGES: activated CD4+ T cells produce cytokines \rightarrow recruit macroPHAGES \rightarrow invade thyroid gland \rightarrow contribute to development of Hashimoto's thyroiditis (type 4 hypersensitivity reaction)
- 53. **Shooting antibody gun**: auto-antibodies against the thyroid gland contribute to development of Hashimoto's thyroiditis (type 2 hypersensitivity reaction)
- 54. "Thyro Global" truck near antibody gun: Hashimoto's thyroiditis often presents with anti-ThyroGlobulin [anti-Tg] antibodies (thyroglobulin: a large precursor produced by thyroid follicular cells)
- 55. "TransPOrt" boxes near antibody gun: Hashimoto's thyroiditis often presents with anti-Thyroid Peroxidase (TPO) antibodies (TPO: an enzyme produced by thyroid follicular cells)
- 56. FAULTY antibody clip leading to FALLING bowtie : Hashimoto's thyroiditis may present with anti-TSH receptor antibodies (BLOCK action of TSH \rightarrow decreased thyroid hormone production)
- 57. **Shot by antibodies & big red bowtie**: Hashimoto's thyroiditis may INITIALLY present with transient HYPERthyroidism (due to release of preformed, stored thyroid hormone during early phase of thyroid gland destruction)
- 58. Large neck mask : Hashimoto's thyroiditis can present with PAINLESS goiter (enlarged thyroid) (elevated TSH \rightarrow hypertrophy of gland)
- 59. **Blue dots surrounding central clearing**: on histology, Hashimoto's thyroiditis displays a lymphocytic infiltrate and germinal centers
- 60. **HURTling over pink-ringed chair**: in Hashimoto's thyroiditis, atrophic follicles are lined with HURTHLE cells (oxyphil cells) (epithelial cells with eosinophilic granular cytoplasm and abundant mitochondria)
- 61. **Fibrous thyroid plant**: Hashimoto's thyroiditis can cause interstitial fibrosis of the gland
- 62. Chess board with B-cell archers: Hashimoto's thyroiditis is the most important risk factor for Primary Lymphoma of the Thyroid (due to chronic inflammation with germinal centers) (a B-cell non-Hodgkin lymphoma)

Endocrine 4.2 - Hypothyroidism: Other Causes





- 1. Untied bowtie by empty iodine case: iodine deficiency is the most common cause of hypothyroidism worldwide (endemic goiter)
- 2. **Empty iodine vial**: iodine deficiency is the most common cause of hypothyroidism worldwide (endemic goiter)
- 3. Falling T4 timebomb: iodine deficiency leads to inadequate production of thyroid hormone (measured as serum T4) (SUBCLINICAL hypothyroidism may be asymptomatic and show a normal T4)
- 4. **Henchman tightening bowtie as T4 bomb falls**: deficiency of thyroid hormone (measured as T4) → increased release of TSH from the pituitary (in an effort to stimulate thyroid hormone production)
- 5. Clenched fist on bartender's collar : iodine deficiency→ thyroid hormone deficiency → increased TSH released from pituitary → hypertrophy and hyperplasia of follicular cells → diffuse, symmetrically enlarged goiter
- 6. **Nodules on face and untied bowtie**: repeated episodes of nodular formation and involution (caused by fluctuating iodine levels) → irregularly enlarged multinodular goiter and by not by roidism
- 7. Angry boss raising T4 timebomb when given iodine: nodules in multinodular goiter may develop the ability to autonomously produce thyroid hormone WITHOUT TSH stimulation→ excess thyroid hormone production and hyperthyroidism after iodine replacement (toxic multinodular goiter)
- 8. **Huffing and puffing**: if goiters become large enough, they can cause compression of the trachea→ dyspnea and stridor
- 9. Race hoarses : if goiters become large enough, they can cause compression of the recurrent laryngeal nerves \to hoarseness
- 10. 10 {\bf Casino Riedel sign}: (Riedel's thyroiditis) an autoimmune, fibrosing thyroiditis leading to hypothyroidism
- 11. **Blue light on thyroid bowtie**: histologically, Riedel's thyroiditis shows an intense inflammatory infiltrate including neutrophils, eosinophils, lymphocytes, and plasma cells
- 12. **Spreading blue lights**: inflammation in Riedel's thyroiditis spreads to involve local muscle, nerves, blood vessels, and adipose tissue
- 13. **Sign throwing cytoCOINS** : release of cytokines leads to fibroblast activation→ fibrosis
- 14. **Fibrotic vines surrounding Casino Riedel sign** : Riedel's thyroiditis is associated with intense fibrosis of the thyroid→ gland effacement and destruction
- 15. **Extensive vine spreading**: fibrosis in Riedel's thyroiditis extends outside thyroid tissue and into surrounding neck structures
- 16. Secret agent Sean covered in leaves : Riedel's thyroiditis is associated with several forms of SYSTEMIC fibrosis (typically as part of IgG4-related systemic diseases) → retroperitoneal fibrosis, autoimmune pancreatitis, sclerosing aortitis
- 17. Four antibody playing cards : Riedel's thyroiditis may be part of a group of IgG4-related systemic diseases (all with elevated levels of IgG-4 antibodies)→ systemic fibrosing lesions rich in plasma cells
- 18. **Diamond neck broach with fibrous neck sash**: Riedel's thyroiditis leads to the formation of a hard, fixed, painless goiter and diffuse fibrosis of surrounding neck structures
- 19. **Ms. Riedel choking player**: diffuse fibrosis of neck structures→ compression of the trachea (dyspnea or stridor), esophagus (dysphagia), or recurrent laryngeal nerves (hoarseness)
- 20. 20
Untied bowtie by Casino Riedel sign : many cases of Riedel's thy
roiditis progress to subclinical and overt hypothyroidism
- 21. Antibody playing cards: two-thirds of patients with Riedel's thyroiditis will have antithyroid autoantibodies (as seen in Hashimoto's thyroiditis), though they do not CAUSE disease in this case (most likely develop in response to self antigens released from destroyed thyroid follicles)

- 22. **Blue chips in cage and covering table**: subacute granulomatous thyroiditis is associated with diffuse, granulomatous inflammation (discreet areas of macrophage aggregation surrounded by helper T cells
- 23. **Multiple pink dice**: multinucleated giant cells (multiple coalesced macrophages) may be seen in subacute granulomatous thyroiditis
- 24. **Middle aged women with cold and antibody cane** : subacute granulomatous thyroiditis is most commonly seen in middle aged women following a viral infection→ autoantibody formation
- 25. **"PLACE YOUR B3T5"**: pathogenesis of chronic granulomatous thyroiditis is thought to involve HLA-B35 polymorphisms and viral infection→ molecular mimicry→ recognition of self antigens in the thyroid→ autoimmune destruction of thyroid
- 26. **Painful blow to red bowtie**: subacute granulomatous thyroiditis presents with an acutely inflamed, PAINFUL goiter
- 27. **Broken glass leaking pink fluid**: acute inflammation in subacute granulomatous thyroiditis causes follicle rupture→ release of stored thyroid hormone
- 28. Henchman with large red bowtie elevating T4 timebomb when struck: thyroid follicle rupture and release of stored thyroid hormone in the acute phase of subacute granulomatous thyroiditis (which can last between 2-8 weeks) can cause transient HYPERthyroidism
- 29. **Beat up henchman with orange untied bowtie**: transient hyperthyroidism in subacute granulomatous thyroiditis is usually followed by a period of transient subacute HYPOthyroidism (usually asymptomatic)
- $30.\,30$ Normal green bowtie \to in most cases of subacute granulomatous thyroiditis, there is complete recovery and a return to a euthyroid state :
- 31. **Silent baby attacking Hashimoto's henchmen**: subacute lymphocytic thyroiditis (silent or painless thyroiditis) is considered to be TRANSIENT variant of chronic autoimmune thyroiditis (Hashimoto's thyroiditis)
- 32. Shooting at THYROGLOBAL and TransPOrt: the early phase of subacute lymphocytic thyroiditis is associated with high titers of anti-thyroglobulin and anti-TPO antibodies
- 33. **Damaging big red bowtie** : the initial inflammation in subacute lymphocytic thyroiditis damages thyroid follicles \rightarrow release of stored thyroid hormone \rightarrow TRANSIENT hyperthyroidism
- 34. Happy baby rattle: subacute lymphocytic thyroiditis usually presents with a PAINLESS goiter
- 35. **Beat up henchman with untied bowtie** : subacute lymphocytic thyroiditis often has a transient period of HYPOthyroidism following an initially HYPERthyroid state (from acute inflammation and follicle rupture→ release of stored thyroid hormone)
- 36. **Normal green bowtie on henchman**: subacute lymphocytic thyroiditis usually has complete resolution and return to a euthyroid state
- 37. **Dress with blue dots and pink patches**: histologically, subacute lymphocytic thyroiditis shows a diffuse lymphocytic infiltrate and the formation of germinal centers (sites of B lymphocyte differentiation
- 38. Secret agent baby: transient silent thyroiditis following pregnancy is seen in about 5% of women within their first postpartum year
- 39. Lithium "LIFT" : lithium can inhibit the release of thyroid hormones→ hypothyroidism and goiter formation (less commonly, lithium can lead to HYPERthyroidism)
- 40. **3 AMIGOS**: amiodarone (a class III antiarrhythmic) blocks the production of T3 and T3 receptors (as well as causing direct toxic injury to the thyroid)
- 41. 41Henchman with TransPOrt briefcase pushed down by iodine wielding henchman: excess iodine directly inhibits thyroid peroxidase, preventing organification of thyroid hormone— HYPOthyroid effect (Wolff-Chaikoff effect

Endocrine 4.3 - Hyperthyroidism: Overview & Graves' Disease





- 1. **Hyper ravenous doberman**: excess thyroid hormone→ hypermetabolic state→ weight loss, increased temperature, flushed skin
- 2. Facing the heat : hypermetabolic state→ sweating and warm, flushed skin from from peripheral vasodilation
- 3. **Tough time getting up**: (thyrotoxic myopathy) hyperthyroidism causes PROXIMAL muscle weakness (hip flexors & quadriceps) WITHOUT elevated creatine kinase or muscle atrophy (as opposed to elevated creatine kinase in hypothyroid myopathy)
- 4. "HOLY SYMPATHY" sign with beta bugles: hyperthyroidism causes signs and symptoms of sympathetic overstimulation (tachycardia, hypertension, diaphoresis) through both a direct effect of thyroid hormone AND increased beta adrenergic receptor expression
- 5. **Shaking scared** : sympathetic overactivity in hyperthyroidism→ anxiety, irritability, tremor (even cognitive impairment and confusion)
- 6. Wide staring gaze : sympathetic activation of the superior tarsal muscles→ widened gaze and lid lag (in which the sclera is visible above the iris on downward gaze)
- 7. Arrhythmia on screen: patients with hyperthyroidism are at an increased risk of cardiac conduction abnormalities, ESPECIALLY atrial fibrillation
- 8. **Steaming ears**: increased cardiac contractility and sympathetic tone in hyperthyroidism→ hypertension
- 10. **Bright cardiac flashlight output** : increased sympathetic activity in hyperthyroidism—increased heart rate and contractility— increased cardiac output (up to 250% in some cases)
- 11. **Swinging reflex hammer**: patients with hyperthyroidism often have brisk, exaggerated deep tendon reflexes
- 12. **Standing in mud puddle**: hyperthyroidism can increase gastrointestinal motility→ diarrhea, malabsorption syndromes, increased weight loss
- 13. Hollowed out tree branch: excess thyroid hormone increases bone resorption → hypercalcemia (hypercalcemia can suppress PTH and vitamin D, increasing the risk of osteoporosis, especially in older women)
- 14. **Lightning bow tie**: (thyroid storm) untreated severe hyperthyroidism can life-threatening thyroid storm (fulminant thyrotoxicosis) due to massive sympathetic stimulation
- 15. Fire suit: (thyroid storm) SEVERE fever is a component of thyroid storm
- 16. Low volume mud puddle : thyroid storm may cause severe gastrointestinal losses from vomiting and diarrhea→ hypovolemia
- 17. Cracked skull bust : thyroid storm may cause CNS dysfunction, ranging from agitation and delirium to stupor and coma
- 18. **Bacterial lanterns over flame suit** : infection may be a precipitating factor for thyroid storm in patients with hyperthyroidism
- 19. **Cast on man near flame suit** : trauma may be a precipitating factor for thyroid storm in patients with hyperthyroidism
- 20. **Scalpel fence posts behind flame suit**: surgery may be a precipitating factor for thyroid storm in patients with hyperthyroidism (both thyroid AND non-thyroid surgery)
- 21. **Elevated bomb trigger, fallen TSH bowtie**: excess thyroid hormone levels in hyperthyroidism exhibit negative feedback on the pituitary, causing LOW TSH levels (the best screening test for hyperthyroidism)
- 22. **Women being lowered into Grave**: Graves' disease (autoimmune hyperthyroidism) is the most common cause of hypothyroidism (more common in women)
- 23. Here LAy B8trice sweet DR3ams: Graves' disease (the most common cause of hyperthyroidism) is highly associated with HLA antigens B8 and DR3

- 24. **Electrifying thyroid antibodies**: the pathogenesis of Graves' disease involves development of ACTIVATING autoantibodies that stimulate TSH receptors→ excess thyroid hormone production
- 25. Large metal neck shackle: chronic stimulation of TSH receptors by autoantibodies in Graves' disease \rightarrow follicular hypertrophy and hyperplasia (even with LOW TSH) \rightarrow diffuse, nontender goiter
- 26. **Protruding goggles** : (exophthalmos) anti-TSH receptor antibodies in Graves' disease activate orbital fibroblasts→ inflammation, fat, and glycosaminoglycan accumulation→ outward bulging of eyes (Graves' ophthalmopathy)
- 27. **Helper holding torch and cytoCOINs**: activation of helper T cells causes orbital inflammation and cytokine release, key components in development of Graves' ophthalmopathy (exophthalmos)
- 28. Glycosaminoglycan GAG and crazy eyes: in Graves' ophthalmopathy (exophthalmos), cytokines released by helper T cells stimulate orbital fibroblasts to produce excess glycosaminoglycans (especially hyaluronic acid) → accumulation in periorbital tissue
- 29. Water accumulating in grave: in Graves' ophthalmopathy (exophthalmos), excess glycosaminoglycans (especially hyaluronic acid) cause water accumulation in periorbital tissues→ outward displacement of eyes
- 30. **Wandering eyes** : swelling of the extraocular muscles in Graves' ophthalmopathy (exophthalmos) can impair their movement→ diplopia
- 31. **Shiny knee high boots**: (infiltrative dermopathy) excess accumulation of glycosaminoglycans in the dermis of the anterior leg→ water accumulation→ waxy edema with dark purple or yellow-brown nodules (pretibial myxedema) (less common finding that Graves' ophthalmopathy)
- 32. Papillary dress pattern: histologically, thyroid tissue in Graves' disease shows diffuse follicular cell hyperplasia, T-cell lymphocytic infiltration, and crowding of the follicles that forms "papillae" projecting into colloid
- 33. **Scalloped white collar** : reduction of colloid in Graves's disease makes it appear to "pull away" from surrounding parenchyma→ "scalloped" appearance of colloid
- 34. Glowing iodine radioactivity: in Graves' disease, the entire thyroid will take up radioactive iodine on scanning, giving it a diffusely "hot" appearance
- 35. **Multinodular face running away with T4 bomb**: tissue in multinodular goiter (a common cause of HYPOthyroidism) can begin to produce thyroid hormone autonomously→hyperthyroidism
- 36. Fallen TSH receptor remote : toxic multinodular goiters produce excess thyroid hormone WITHOUT stimulation by TSH→ hyperthyroidism (high T4, LOW TSH)
- 37. **Scattered glowing iodine**: toxic multinodular goiters will show discrete areas of functional "hot" nodule radioactive iodine uptake (unlike diffuse uptake in Graves' disease)
- 38. Cancer crab gravestone : nodules that do not take up radioiodine on scanning ("cold" nodules) are much more likely to be cancerous that "hot" nodules
- 39. **Glowing helmet with plus sign**: toxic adenomas are solitary nodules that autonomously produce thyroid hormone WITHOUT the need for TSH→ hyperthyroidism
- 40. **Elevated T4 bomb with fallen TSH trigger**: toxic adenomas often have an ACTIVATING TSH receptor mutation→ autonomous production of thyroid hormone→ hyperthyroidism (high T4, LOW TSH)
- 41. **Smooth white helmet on jetpack henchman**: toxic thyroid adenomas are made of follicular cells that produce thyroid hormone (functional follicular adenoma) and are surrounded an intact capsule (invasion of the capsule → follicular CARCINOMA)
- 42. Glowing iodine taken by henchman: toxic adenomas are take up radioactive iodine, giving the a "hot" appearance (a feature of benign thyroid nodules)

Endocrine 4.4 - Thyroid Nodules & Cancer





- 1. **Multinodular face and untied bowtie**: multinodular goiter is the most common thyroid nodularity (secondary to iodine deficiency hypothyroidism
- 2. **Disarming radioactive glow**: nodules that take up radioactive iodine ("hot" nodules) are rarely malignant (though they may produce excess thyroid hormone (toxic nodule) hyperthyroidism)
- 3. "DUE FOR INSPECTION" mine: nodules that do not take up radioactive iodine ("cold" nodules) should be biopsied to rule out malignancy
- 4. Bowtie balloon: most SOLITARY thyroid nodules are benign, fluid-filled cysts
- 5. **Balloon bowtie on fire** : inflammation (as with Hashimoto's or infectious thyroiditis) can produce focal areas of enlargement→ nodule formation
- 6. **ADDed protection of helmet** : follicular adenomas are BENIGN neoplastic nodules that RARELY produce thyroid hormone
- 7. **Follicular net**: follicular adenomas are composed of sheets of follicular cells surrounding colloid (similar to normal thyroid tissue)
- 8. Intact helmet encapsulating head : follicular adenomas have a well defined, INTACT CAPSULE (invasion of capsule→ follicular carcinoma)
- 9. Radiation suit : almost all thyroid malignancies are associated with a history of radiation to the neck
- 10. **HenchMAN kidnapping child on crab jet ski** : thyroid masses are much more likely to be malignant in males and children
- 11. Woman waving #1 finger: papillary thyroid carcinoma is the most common thyroid malignancy (up to 85% of cases, most in adult women)
- 12. Radioactive boat decal: papillary thyroid carcinoma has the strongest association with radiation
- 13. **Activating onboard map**: most cases of papillary thyroid carcinoma are due to activating mutations of the mitogen activated protein kinase (MAPK) signalling
- 14. NeTwoRK1 signal: NRTK1 tyrosine kinase mutations lead to MAPK activation
- 15. **RETurn if found briefcase**: RET tyrosine kinase mutations lead to MAPK activation
- 16. **Benjamin Franklin bills flying out** : B-raf mutations are the most common cause of MAPK activation→ unregulated cell proliferation
- 17. Cancer crab on buoy: papillary carcinoma may be solitary or metastasize to cervical lymph nodes
- 18. Purple finger algae on propellor: papillary thyroid carcinoma forms finger like projections and typically LACK colloid or follicles
- 19. **Open air sea breeze boat** : papillary thyroid carcinomas are typically unencapsulated
- 20. Rose pattern on dress: (psammoma bodies) dystrophic calcification with swirled eosinophilic appearance present in about half of papillary thyroid carcinomas
- 21. Hollow white goggles→ papillary thyroid carcinoma cell nuclei have a clear, "Orphan Annie eye" appearance due to very fine chromatin :
- 22. Mature female pilot towing follicular net: follicular thyroid carcinoma is most common in older women and is the second most common thyroid malignancy
- 23. **Iodine tank leak** : follicular thyroid carcinoma is more common in iodine deficient areas

- 24. RAS rat : many cases of follicular thyroid carcinoma are to due activation of the RAS oncogene→ uninhibited cell growth
- 25. **Cracked glass enclosure**: capsular invasion is diagnostic of follicular thyroid CARCINOMA (as opposed to follicular adenoma with an INTACT capsule)
- 27. **Old woman in plastic suit** : anaplastic thyroid carcinoma most commonly occurs in the elderly (>65 years)
- 28. Kill gesture: anaplastic thyroid carcinoma has a nearly 100% mortality rate
- 29. **Boat and helicopter guards**: anaplastic thyroid carcinoma can arise sporadically or from progression of papillary or follicular carcinoma
- 30. **Broken railroad crossing arm**: anaplastic thyroid carcinoma is associated with loss of p53 cell division checkpoint regulation
- 31. Choked cyanotic henchman : anaplastic thyroid carcinoma grows aggressively into surround tissue \to tracheal compression
- 32. **Pleomorphic cracks in skull base**: anaplastic thyroid carcinoma cell nuclei are often large and pleomorphic or spindle shaped
- 33. **Mitotic skull eyes**: anaplastic thyroid carcinoma has prominent mitotic figures, areas of necrosis, and capsular invasion on histology
- 34. Sea "C" shells : medullary thyroid carcinomas arise from parafollicular C cells
- 35. **Exposed outer wiring**: parafollicular C cells are NEUROENDOCRINE cells lining the periphery of thyroid follicles
- 36. **Agents A and B**: most cases of medullary thyroid carcinoma are sporadic, but up to 20% are associated with MEN2A and MEN2B
- 37. "Point of no RETurn": most medullary thyroid carcinomas are associated with activating mutations of the RET protooncogene (tyrosine kinase)
- 38. Cancer crab on string of buoys: medullary thyroid carcinoma may be solitary or metastasize to cervial lymph nodes
- 39. Tone of C speaker: medullary thyroid carcinomas may produce carcinoma due
- to their C cell origin (calcitonin can be used as a tumor marker)

 40. **Mud leaking from door**: medullary thyroid carcinomas may produce
- calcitonin→ increased gut motility or decreased intestinal fluid absorption→ diarrhea
 41. "VIP only" door under moon and smiley face : neuroendocrine tumors like
- 41. "VIP only" door under moon and smiley face: neuroendocrine tumors like medullary thyroid carcinoma may produce several hormones (VIP→ diarrhea, serotonin→ flushing, ACTH→ Cushing's syndrome
- 42. Bleeding skull tower: medullary thyroid carcinomas are usually solid with prominent areas of necrosis and hemorrhage
- 43. **Spindle shell reef**: medullary thyroid carcinoma cells form nests of polygonal or spindle shaped cells
- 44. Dark spots on electrical box : medullary thyroid carcinomas are composed of neuroendocrine cells \rightarrow dense secretory granules in cytoplasm
- 45. **Armored amyloid lady in pink puddles**: calcitonin can precipitate within medullary thyroid carcinomas to form amyloid deposits
- 46. Flashing red and green lights: amyloid deposits (precipitated calcitonin) in medullary thyroid carcinoma appear red (normal light) or green (polarized light) with Congo red staining

Endocrine 4.5 - Parathyroid Gland Disorders





- 1. 4 buttons with thyroid bowtie & PthD jacket: the 4 parathyroid glands (located adjacent to the thyroid) secrete parathyroid hormone (PTH)
- 2. **PthD at #1 ice cream station**: 1º (primary) HYPERparathyroidism (PARATHYROID glands secrete excessive PTH)
- 3. Female scientist: 1º HYPERparathyroidism is most common in older women
- 4. **Oversized Calci-Yum ice cream**: HYPERparathyroidism causes increased free calcium levels (ionized calcium) (due to excessive PTH)
- 5. "ADD toppings": parathyroid ADENOMA (most common cause of 1° HYPERparathyroidism) (usually sporadic & involves a single gland)
- 6. "Agent P": multiple endocrine neoplasia 1 (MEN1) can cause 1º HYPERparathyroidism (familial)
- 7. "Agent A": MEN2A can can cause 1º HYPERparathyroidism (familial)
- 8. "Agent P" closest to "ADD" poster : in MEN1, 1º HYPERparathyroidism is most commonly due to a parathyroid ADENOMA
- 9. "Agent A" closest to oversized buttons: in MEN2A, 1° HYPERparathyroidism is most commonly due to parathyroid HYPERPLASIA
- 10. "CHIEF" PthD: PTH is secreted by CHIEF cells in the parathyroid glands
- 11. "CHIEF" badge taking over ONE button : parathyroid ADENOMAS are composed of a focal clusters of chief cells (usually in a single gland) \rightarrow hypersecretion of PTH
- 12. **Dome encapsulating purple sprinkles**: parathyroid ADENOMAS display well-encapsulated sheets of purple CHIEF cells
- 13. **3 TINY buttons stained with ice cream splotches**: in parathyroid ADENOMAS, the 3 normal parathyroid glands undergo ATROPHY (adenoma secretes excessive PTH → hypercalcemia → suppression of 3 normal glands
- 14. Oversized buttons : parathyroid HYPERPLASIA can cause 1 $^{\circ}$ HYPERparathyroidism (enlargement of all 4 glands \rightarrow diffuse hypersecretion of PTH by chief cells)
- 15. CRAB spoon spilling ice cream & purple sprinkles: primary parathyroid CARCINOMA can cause 1º HYPERparathyroidism (extends via local invasion or metastasis)
- 16. **CALCITRON D3 robot**: PTH leads to increased synthesis of CALCITRIOL (1,25-dihydroxyvitamin D) (by increasing activity of 1-alpha-hydroxylase in the kidney
- 17. CALCITRON robot retrieving Calci-Yum from gut fridge: calcitriol increases CALCIUM absorption from the GI system
- 18. **CALCITRON robot retrieving "P" mixer from gut fridge** : calcitriol increases PHOSPHATE absorption from the GI system
- 19. Kidney mixer: PTH exerts direct effects on the kidney
- 20. Fallen "P" mixer: elevated PTH leads to HYPOphosphatemia (by increasing phosphate EXCRETION in the kidney proximal tubule)
- 21. Raised Calci-yum ice cream: elevated PTH leads to HYPERcalcemia (by increasing calcium RESORPTION in the kidney proximal tubule)

- 22. Excited CLASSmates : in 1 0 HYPERparathyroidism , elevated PTH leads to activation of osteoCLASTS \rightarrow bone RESORPTION
- 23. Bone card punched with CYSTIC holes : ELEVATED PTH can present with OSTEITIS FIBROSA CYSTICA (over-active osteoCLASTS \rightarrow excessive bone resorption)
- 24. **BROWN punched out pieces**: OSTEITIS FIBROSA CYSTICA can present with BROWN tumors (collections of osteoCLASTS mixed with fibrous tissue and poorly mineralized bone)
- 25. Cleaning SUB-tablecloth: ELEVATED PTH can cause SUBperiosteal thinning of cortical bone (due to excessive bone resorption)
- 26. **Torn finger gloves**: ELEVATED PTH may cause SUBperiosteal thinning of the phalanges
- 27. **Torn upper chest**: ELEVATED PTH may cause SUBperiosteal thinning of the clavicles
- 28. **Speckled hair**: ELEVATED PTH may cause a "salt and pepper" appearance of the skull (due to SUBperiosteal thinning)
- 29. Raised chalk: ELEVATED PTH leads to elevated alkaline phosphatase (due to excessive bone resorption)
- 30. **Thin bone breaking**: ELEVATED PTH can lead to low bone mineral density or osteoporosis (due to excessive bone resorption)
- 31. **2** raised yellow test tubes: ELEVATED PTH can cause type II renal tubular acidosis (non-anion gap metabolic acidosis) by impairing bicarbonate reabsorption in the proximal tubule
- 32. **STONE obstructing kidney tray**: HYPERCALCEMIA can cause calcium stones (due to hypercalciURIA
- 33. Excessively watery crotch: HYPERCALCEMIA can cause polyuria (excessive urination) due to defects in renal concentrating ability
- 34. Actively attempting to drink water: HYPERCALCEMIA can cause polydipsia (excessive thirst) due to defects in renal concentrating ability
- 35. Kidney tray with Calci-Yum samples : severe chronic HYPERCALCEMIA can cause metastatic calcification of the renal tubules (\rightarrow end stage renal disease)
- 36. **Shirt with Calci-yum bits**: severe chronic HYPERCALCEMIA can cause metastatic calcification (calcium deposits in lungs, brain, and skin
- 37. **Brandishing plunger and clutching stomach**: HYPERCALCEMIA can cause constipation (due to decreased smooth muscle tone and abnormal autonomic function)
- 38. Acidic lemon water leading to floor erosion: HYPERCALCEMIA can cause peptic ulcer disease (excessive calcium \rightarrow stimulates gastrin release in the stomach \rightarrow increased secretion of gastric acid \rightarrow ulcer formation)
- 39. Squeezing pancreatic sponge to clean ice cream: HYPERCALCEMIA can cause pancreatitis (due to calcium deposition or activation of trypsinogen)
- 40. **Mad scientist with crazy eyes**: HYPERCALCEMIA can cause psychiatric "overtones" (confusion, anxiety, psychosis, seizures)

Endocrine 4.5 - Parathyroid Gland Disorders





- 41. "99 dessert TECHNIQUES": a TECHNETIUM-99m-sestamibi radionuclide scan be used to localize hyperfunctioning parathyroid tissue (causing 1° HYPERparathyroidism) prior to surgery
- 42. **Scalpel picture**: symptomatic 1º HYPERparathyroidism is treated with surgery
- 43. Osteo BUILDER installing bone bookcase: surgical removal of hyperfunctioning parathyroid causes rapid increase in bone mineralization (abrupt decrease in PTH → increase in osteoBLAST activity) ("hungry bone syndrome")
- 44. **Dropped Calci-Yum, phosphate mixer & magnesium magazine**: in hungry bone syndrome, rapid increase in bone mineralization → abrupt decrease in calcium, phosphate, and magnesium levels (due to rapid increase in bone mineralization)
- 45. PthD at #2 ice cream station : 2° (secondary) HYPERparathyroidism (due to excessive PTH secretion by the KIDNEYS)
- 46. **Small crumpled kidney cups** : chronic kidney disease (CKD) causes 2º HYPERparathyroidism
- 47. **Malfunctioning CALCITRON robot**: CKD leads to decreased synthesis of CALCITRIOL (Vitamin D) (due to 1-alpha-hydroxylase deficiency)
- 48. **Dripping Calci-Yum ice cream**: 2^o HYPERparathyroidism presents with HYPOcalcemia (CKD \rightarrow decreased calcitriol \rightarrow decreased calcium absorption from GI tract)
- 49. **PthHD**: 2^o HYPERparathyroidism presents with increased PTH (decreased calcitriol & decreased calcium \rightarrow each stimulate secretion of PTH by the parathyroid glands)
- 50. "P" mixers raised toward PthD jacket : 2° HYPERparathyroidism presents with HYPERphosphatemia (CKD \rightarrow increased retention of phosphate in the kidney) \rightarrow stimulates secretion of PTH by the parathyroid gland)
- 51. **4 OVERSIZED buttons**: 2º HYPERparathyroidism presents with diffuse parathyroid HYPERPLASIA (due to chronic stimulation of parathyroids by low calcium, low calcitriol, and high phosphate)
- 52. Bone card punched with cystic holes : 2^{o} HYPERparathyroidism can cause osteitis fibrosa cystica (elevated PTH \rightarrow excessive bone resorption)
- 53. **Raised chalk**: 2º HYPERparathyroidism leads to elevated alkaline phosphatase (elevated PTH → excessive bone resorption)
- 54. PthD at #3 ice cream station : 3° (tertiary) HYPERparathyroidism (a result of long-standing 2° HYPERparathyroidism)
- 55. **Small crumpled kidney cups**: chronic kidney disease (CKD) is the initiating factor in 3° HYPERparathyroidism
- 56. 4 massive buttons: 3º HYPERparathyroidism presents with marked nodular HYPERPLASIA of parathyroid glands (due to persistent stimulation by low calcium, low calcitriol, and high phosphate)
- 57. **Overly raised PthD**: 3º HYPERparathyroidism presents with markedly elevated PTH (hyperplastic parathyroid glands secrete PTH autonomously [regardless of serum calcium levels])

- 58. **Raised Calci-Yum**: 3º HYPERparathyroidism presents with HYPERcalcemia (elevated PTH → increased bone resorption)
- 59. **Raised "P" mixer**: 3º HYPERparathyroidism presents with HYPERphosphatemia (CKD → increased retention of phosphate in the kidney) (also elevated PTH → increased bone resorption)
- 60. **Severed CALCITRON robot head** : 3º HYPERparathyroidism presents with LOW calcitriol (Vitamin D) (persistent CKD → persistent 1-alphahydroxylase deficiency)
- 61. Littered with Calci-Yum bits: in 3° HYPERparathyroidism, severe HYPERcalcemia can cause cause metastatic calcification (calcium deposits in lungs, brain, and skin)
- 62. **Brandishing scalpel**: 3º HYPERparathyroidism is treated with surgery (parathyroidectomy)
- 63. **Torn discarded PthH coat**: HYPOparathyroidism (low PTH secretion by parathyroid glands)
- 64. **Scalpel hook**: surgical injury to the parathyroid glands is the most common cause of HYPOparathyroidism (thyroid or parathyroid surgery)
- 65. **Radiation microwave**: head and neck radiation can cause HYPOparathyroidism
- 66. **Antibody hooks with gland-y hats**: type 1 autoimmune polyglandular syndrome can cause HYPOparathyroidism (as well as adrenal insufficiency & injury to other endocrine organs)
- 67. **George Washington with empty Calci-Yum scoop**: DiGeorge syndrome causes HYPOparathyroidism (congenital 22q11 deletion) (results from abnormal development of 3rd and 4th branchial pouches)
- 68. **Hiding "P" mixers**: HYPOparathyroidism presents with HYPERphosphatemia (decreased PTH → decreased phosphate excretion in kidneys)
- 69. **Spilled Calci-Yum bowl**: HYPOparathyroidism causes HYPOcalcemia (PTH → decreased calcium reabsorption in kidneys, decreased bone resorption, decreased calcitriol)
- 70. White lips, hands & feet: mild HYPOCALCEMIA can cause perioral numbness & paresthesias of hands and feet
- 71. **Stiff shaking leg**: severe HYPOCALCEMIA can cause tetany (muscle cramps or severe spasms)
- 72. Face taps : HYPOCALCEMIA can cause Chvostek's sign (tapping of facial nerve \rightarrow facial twitching)
- 73. Exerting arm pressure : HYPOCALCEMIA can cause Trousseau's sign (occlusion of brachial artery with BP cuff → hand spasms)
- 74. "SEIZE THE DAY": severe HYPOCALCEMIA can cause seizures
- 75. **Floppy heart balloon**: severe HYPOCALCEMIA can cause heart failure, hypotension, and shock (low calcium impairs contractility of heart and arterial tone)

Endocrine 4.6 - Multiple Endocrine Neoplasia (MEN)



- 1. **MEN** in slacks: Multiple endocrine neoplasia (MEN) (inherited disorder characterized by tumor formation in multiple endocrine organs)
- 2. **Family**: family history of endocrine tumors is a risk factor for MEN
- 3. **BILATERAL crab signs**: MEN presents with multiple tumors (sometimes bilateral)
- 4. Young kid: in MEN, endocrine tumors occur at a young age (< age 35)
- 5. "Welcome back": in MEN, tumors frequently recur (even after surgical removal)
- 6. Domino barrier: all forms of MEN are autosomal dominant
- 7. Head agent "P": MEN type 1 (due to mutations in the MEN1 gene)
- 8. "11" antennae: the MEN1 gene is located on chromosome 11
- 9. Cancer-fighting ribbon: MEN1 is a tumor suppressor gene
- 10. "Paranormal Population Police": MEN1 is associated with tumors in the Parathyroid, Pancreas, and Pituitary
- 11. **4 neck buttons**: MEN1 is associated with PARATHYROID tumors (usually involving more than one parathyroid gland)
- 12. "**PthD**": in MEN1, PARATHYROID tumors cause primary HYPERPARATHYROIDISM (presents with increased levels of parathyroid hormone [PTH])
- 13. **Holding up Calci-YUM ice cream**: in MEN1, primary HYPERPARATHYROIDISM presents with elevated serum calcium (identical to presentation of sporadic primary hyperparathyroidism)
- 14. Pancreatic sponge: MEN1 is associated with PANCREATIC tumors
- 15. **Multiple ISLANDS on Hawaiian shirt**: in MEN1, PANCREATIC tumors are primarily of the ISLET cell type
- 16. "GAS": in MEN1, GASTRINOMAS are the most common PANCREATIC tumor
- 17. **Asphalt erosion**: GASTRINOMAS cause peptic ulcer disease (due to elevated gastrin levels)
- 18. **Skull buckle over Hawaiian shirt**: in MEN1, PANCREATIC tumors are the #1 cause of death
- 19. **Pituitary diaper bag**: MEN1 is associated with PITUITARY tumors (can be composed of any anterior pituitary cell line)
- 20. **Baby bottles**: in MEN1, PROLACTINOMAS (lactotroph adenomas) are the most common PITUITARY tumor (identical to sporadic prolactinomas)

- 21. Junior Agent "A": MEN type 2A
- 22. Junior Agent "B": MEN type 2B
- 23. "RETurn to your planet": MEN2A and 2B are caused by a gain-offunction mutation in the RET gene
- 24. 10 fingers: the RET gene is located on chromosome 10
- 25. Tire swing: RET encodes a receptor tyrosine kinase
- 26. **Neck bowtie on crab alien**: MEN2A and 2B are associated with MEDULLARY THYROID cancer
- 27. "C" tiles around sand follicles: MEDULLARY THYROID cancer is a malignancy of the parafollicular C cells)
- 28. **Overgrowth of "C" tiles**: in MEN2A and 2B, MEDULLARY THYROID cancer is preceded by C cell hyperplasia (diffuse, bilateral) (different from sporadic medullary thyroid cancer)
- 29. "Frozen Color" shaved ice: MEN2A and 2B are associated with PHEOCHROMOCYTOMAS
- 30. Adrenal toppings: PHEOCHROMOCYTOMA is a tumor of the adrenal medulla (similar manifestations in MEN2 and sporadic forms)
- 31. **4 neck buttons next to Agent "A"**: MEN2A is associated with PARATHYROID hyperplasia
- 32. "PthD" next to Agent "A": in MEN2A, PARATHYROID hyperplasia causes primary HYPERPARATHYROIDISM (presents with increased levels of parathyroid hormone [PTH])
- 33. **Lip bumps on Agent "B"**: MEN2B presents with MUCOSAL NEUROMAS (small collections of nervous tissue on tongue, lips, and buccal mucosa)
- 34. **Brown puddle & plunger next to Agent "B"**: MEN2B can present with constipation, diarrhea, or other GI abnormalities (due to diffuse INTESTINAL GANGLIONEUROMATOSIS)
- 35. 35Lanky Agent "B" martian: MEN2B can present with "Marfanoid" habitus (lordosis, kyphosis, lengthening of long bones)

Reproductive & GU 1.1 - Abnormal Uterine Bleeding (AUB) & Endometriosis





- 1. Fertile uterine soil: endometrium (epithelium)
- 2. Smooth muscle tile path: myometrium (smooth muscle)
- PALM COEIN fountain: abnormal uterine bleeding can be due to structural problems (Polyps, Adenomyosis, Leiomyomas, Malignancy/Hyperplasia) and non-structural problems (Coagulopathy, Ovulatory, Endometrial, latrogenic, Not otherwise specified)
- Polypy beets: endometrial polyps (hyperplastic growth of endometrial glands and stroma) can cause abnormal uterine bleeding
- 5. **Spots of beet juice** : endometrial polyps can cause light intermenstrual bleeding (spotting)
- 6. Small crab roots : endometrial polyps have a small risk of underlying endometrial cancer
- 7. **ADDING dirt to the myometrial path**: in adenomyosis, the stratum basalis layer of the endometrium grows into the myometrium
- 8. Enlarged globular pumpkins : adenomyosis causes myometrial hypertrophy ightharpoonup uniformly enlarged globular uterus
- 9. Leaking pumpkin: adenomyosis causes heavy prolonged menstrual bleeding
- 10. Pain in abdomen: adenomyosis presents with dysmenorrhea (painful menses)
- 11. Pain in abdomen: adenomyosis presents with dysmenorrhea (painful menses)
- 12. Black female on tiles: fibroids are most common in black women
- 13. Female symbols : fibroids are estrogen-sensitive
- 14. **Pregnant lady laying down tiles**: fibroids increase in size during pregnancy (estrogen sensitive) and decrease in size after menopause
- 15. Inner bulging myometrial tiles : fibroids may be located near the inner endometrial surface (submucosal)
- 16. Outer bulging myometrial tiles: fibroids may be located underneath the outermost serosal surface of the uterus (subserosal)
- 17. Middle bulging myometrial tiles: fibroids may be located in the middle of the uterine wall (intramural)
- 18. Pooling red sap : submucosal fibroids can cause heavy menstrual bleeding
- 19. Barren tree : submucosal fibroids can cause infertility
- 20. Obstruction of colonic plant cover : large posterior fibroids can compress the colon or rectum \rightarrow obstructive symptoms (e.g. constipation)
- 21. **Knocking over bladder can**: large anterior fibroids (or posterior fibroids causing the uterus to displace upward) can cause obstructive urinary symptoms (urgency or incomplete emptying)
- 22. Irregular proliferating grass : endometrial hyperplasia (abnormal proliferation of endometrial cells)
- 23. **ESTRO-GROW** fertilizer: endometrial hyperplasia can be caused by exposure to unopposed estrogen (relative to progesterone) (e.g. estrogen replacement therapy, tamoxifen use, estrogen secreting tumor, anovulatory cycles)
- 24. "TM": tamoxifen (SERM used in treatment of breast cancer) is a risk factor for endometrial hyperplasia (due to agonist effect at estrogen receptors in the uterus)
- 25. Obese gardner: obesity is a risk factor for endometrial hyperplasia (due to increased aromatization of androgens to estrogen in adipose tissue)
- 26. **Unruptured polycystic buds**: polycystic ovarian syndrome (PCOS) is a risk factor for endometrial hyperplasia (due to anovulatory cycles)
- 27. Early bloomers : early menarche is a risk factor for endometrial hyperplasia (due to anovulatory cycles)

- 28. Evergreens/Late deciduous: late menopause is a risk factor for endometrial hyperplasia (due to anovulatory cycles
- 29. **Seedless** : nulliparity is a risk factor for endometrial hyperplasia (due to an ovulatory cycles)
- 30. **L-inch worm**: Lynch syndrome (hereditary nonpolyposis colorectal cancer - HNPCC) is a risk factor for endometrial hyperplasia
- 31. **OCP lawnmower** : oral contraceptive pills decrease the risk of endometrial hyperplasia (due to presence of both progestins and estrogen)
- 32. Leaking puddle: in premenopausal women, endometrial hyperplasia can present with heavy menstrual bleeding
- 33. **Trailing spots**: in premenopausal women, endometrial hyperplasia can present with light intermenstrual bleeding (spotting)
- 34. **Old woman with spotted pants**: endometrial hyperplasia can present with postmenopausal bleeding
- 35. Simple hyperplasia grass : endometrial carcinoma may be simple (increased number of glands), with or without atypia
- 36. Complex hyperplasia grass: endometrial carcinoma may be complex (crowded glands), with or without atypia
- 37. ATYPICAL: the presence of atypia (dysplastic epithelial cells) confers a higher rate of progression to endometrial carcinoma
- 38. Invasive endometrial crabgrass: endometrial carcinoma is usually caused by exposure to unopposed estrogen (relative to progesterone), and has the same risk factors as endometrial hyperplasia
- 39. Extra-uterine endometrial soil: in endometriosis, endometrial glands and stroma are located outside the uterus
- 40. **Throwing endometrial dirt**: reverse menses through the fallopian tube is a hypothesis for formation of extrauterine endometrial tissue (endometriosis)
- 41. Coelomic compost pile: coelomic metaplasia (inappropriate differentiation of pluripotent stem cells) is a hypothesis for formation of extrauterine endometrial tissue (endometriosis)
- 42. **Red sprinkler lines**: vascular and lymphatic spread is a hypothesis for formation of extrauterine endometrial tissue (endometriosis)
- 43. Pile of dirt on ovarian patio: endometriosis forms endometriomas ("chocolate cysts") in the ovaries (most common site)
- 44. **Dirt on intestinal tunnel**: the serosal surface of the intestines is a site of implantation for endometriosis
- 45. Dirt on Fallopian path: the fallopian tubes are a site of implantation for endometriosis
- 46. Dirt in Douglas's pouch : the rectal pouch of Douglas (space between rectum and uterus) is a site of implantation for endometriosis
- 47. **Abdomen hit with dirt**: endometriosis presents with pain (dysmenorrhea, dyspareunia), not abnormal bleeding
- 48. Painful poop pile: endometriosis can present with dyschezia
- 49. Barren tree : endometriosis can cause infertility
- 50. **Dirt powder spots, pink nodules, and creepers**: endometriosis appears as "powder burn" lesions, flesh colored nodules, and filmy adhesions on serosal surfaces
- 51. OCP leaf blower: endometriosis can be treated with oral contraceptive pills (OCPs)

Reproductive & GU 1.2 - Cervical Neoplasia

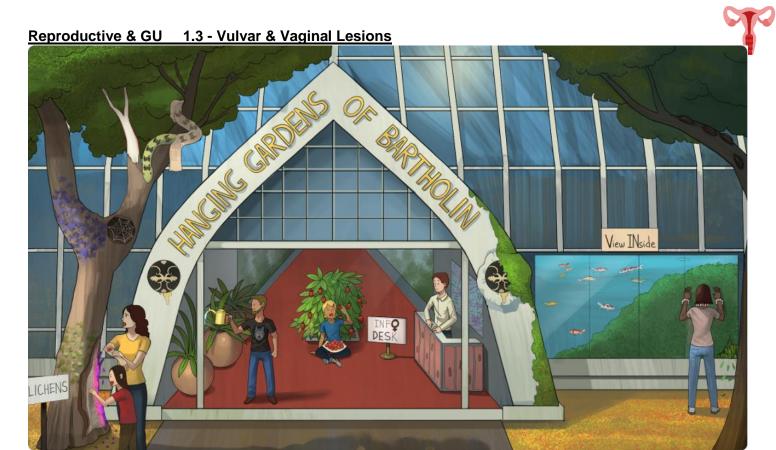




- 1. Stairs to uterine platform: INTERNAL OS of the cervix (opening of cervical canal into uterus)
- 2. Stairs to vaginal walkway: EXTERNAL OS of the cervix (opening of cervical canal into vagina)
- 3. ENDOcervical wall: ENDOcervix (area of cervix lining CERVICAL CANAL)
- 4. EXOcervical wall: EXOcervix (area of cervix exposed to VAGINA)
- 5. COLUMNAR caterpillars : ENDOCERVIX is lined by mucus-secreting COLUMNAR cells
- 6. **SQUAMOUS butterflies**: EXOCERVIX is lined by stratified SQUAMOUS cells
- 7. Polpy fruit : ENDOCERVICAL POLYPS (benign polyps arising from ENDOcervix)
- 8. COLUMNAR caterpillar : ENDOCERVICAL POLYPS are lined by mucussecreting COLUMNAR cells (similar to endocervix)
- 9. **Middle-aged woman with kids**: ENDOCERVICAL POLYPS are most common in middle-aged women with history of multiple pregnancies
- 10. Leaking red fruit: ENDOCERVICAL POLYPS cause VAGINAL BLEEDING
- 11. Caterpillar to butterfly TRANSITION: SQUAMO-COLUMNAR JUNCTION ("TRANSFORMATION zone") (columnar epithelium meets squamous epithelium)
- 12. Purple stamens on milky white flowers : LACTOBACILLI (gram positive rods) in the vagina produce LACTIC ACID ightarrow acidic pH (3.8-4.5)
- 13. **METAmorphosis** at **TRANSFORMATION** zone: at the transformation zone, endocervical columnar cells undergo benign SQUAMOUS METAPLASIA (due to acidic pH in the vagina)
- 14. **ABNORMAL dead butterflies** : squamous metaplasia (a normal process) can progress to DYSPLASIA (cervical intraepithelial neoplasia [CIN])
- 15. $\mbox{\bf PILL bugs}$: cervical dysplasia (CIN) and cancer are caused by human papilloma virus (HPV)
- 16. Car keys: HPV 16 is a HIGH RISK HPV strain (causes dysplasia and cancer)
- 17. "I voted": HPV 18 is a HIGH RISK HPV strain (causes dysplasia and cancer)
- 18. **Broken CHECKPOINT** : p53 tumor suppressor gene (CHECKPOINT of cell cycle) is INHIBITED by high risk HPV \rightarrow uncontrolled cellular replication \rightarrow dysplasia and cancer
- 19. **ROOT BEER**: Rb tumor suppressor gene (break on cell cycle) is INHIBITED by high risk HPV \rightarrow uncontrolled cellular replication \rightarrow dysplasia and cancer
- 20. buG room 1 \rightarrow Solarium : p53 and Rb tumor suppressor genes function at G1 to S phase TRANSITION
- 21. "6" on checkpoint : high risk HPV strains integrate into host DNA \to overexpression of oncogene E6 \to inhibition of p53
- 22. **7 straw in root beer** : high risk HPV strains integrate into host DNA \to overexpression of oncogene E7 \to inhibition of Rb
- 23. **Kissing couple**: multiple HIGH RISK PARTNERS and unprotected intercourse are risk factors for CIN and cancer (due to increased HPV risk)
- 24. $\mbox{\bf Crutches}: \mbox{IMMUNODEFICIENCY}$ is a risk factor for CIN and cancer
- 25. Smoker: SMOKING is a risk factor for CIN and cancer
- 26. **Pill bugs at the transformation zone**: HPV has an affinity for the IMMATURE METAPLASTIC SQUAMOUS cells at the transformation zone

- 27. KOI fish eating pill bugs : on histology, HPV causes KOILOCYTOSIS within squamous epithelial cells
- 28. **Big-eyed KOI fish**: koilocytosis presents as PERINUCLEAR CLEARING ("perinuclear vacuolization") within squamous cells

 29. **ABNORMAL dead butterflies**: HPV can cause DYSPLASIA (cervical
- intraepithelial neoplasia [CIN])
- 30. "C-Inside" for ABNORMAL dead butterflies : cervical dysplasia (CIN) arises from the BASAL epithelial cells and has 3 classes (CIN 1, 2, or 3)
- 31. **ONE-THIRD abnormal butterflies**: CIN 1 (MILD dysplasia involving lower ONE-THIRD of epithelium)
- 32. **TWO-THIRD abnormal butterflies**: CIN 2 (MODERATE dysplasia involving lower TWO-THIRDs of epithelium)
- 33. **Greater than TWO-THIRDS abnormal butterflies**: CIN 3 (SEVERE dysplasia involving MORE THAN TWO-THIRDS or full thickness of epithelium)
- 34. **Breaking through bottom of glass**: CIN can progress to invasive cervical carcinoma (invasion of BASEMENT MEMBRANE)
- 35. Low SIL: LSIL (LOW-GRADE squamous intraepithelial lesion) (alternate term for CIN 1
- 36. **Pool filter clearing butterflies**: CIN1 (aka LSIL) usually RESOLVES spontaneously in young healthy women
- 37. **High SIL**: HSIL (HIGH-GRADE squamous intraepithelial lesion) (alternate term for CIN 2 and 3)
- 38. Filter clogged with butterflies : CIN2/3 (aka HSIL) are at higher risk of progressing to cervical CANCER
- 39. **Brushing butterflies**: PAP SMEAR (screening test for cervical DYSPLASIA and CANCER (collected by taking a swab of the cervix) (important to sample transformation zone)
- 40. **ABNORMAL butterflies detected**: if abnormal cells are detected by Pap smear, colposcopy and biopsies are performed
- 41. Scope: COLPOSCOPY is performed by visualizing the cervix with a scope
- 42. **Pool tester turning water WHITE** : during colposcopy, ACETIC ACID is applied to the cervix to turn abnormal areas WHITE
- ${\bf 43. \; Grabber \; tool}: {\bf during \; colposcopy}, \, {\bf abnormal \; tissue \; is \; BIOPSIED}$
- 44. Cancer crabs : cervical cancer is most commonly SQUAMOUS CELL carcinoma (less commonly adenocarcinoma)
- 45. **PEARL necklace**: SQUAMOUS CELL carcinoma of the cervix has keratin PEARLS and intercellular bridges
- 46. **Pinching ureteral hose** : cervical cancer spreads down and out \rightarrow compression of URETERS \rightarrow postrenal kidney injury
- 47. Leaking red post-pollination : cervical cancer may present with POSTCOITAL BLEEDING
- 48. **Protective syringe fence**: HPV VACCINE protects against common HPV types (including low and high risk strains)



- 1. Secreting duct at "Garden's of Bartholin": Bartholin glands are secretory glands that drain via ducts in the vulva
- 2. **Bulging gunky air duct**: blockage of a Bartholin duct causes the gland to dilate with fluid → Bartholin cyst (forms Bartholin abscess if infected)
- 3. "LICHENS": vulvar lichen sclerosus, lichen planus, and lichen simplex chronicus are benign skin conditions
- 4. **Itchy skin**: the vulvar "lichens" all present with severe pruritus and visual skin changes
- 5. Sclerotic snake: lichen SCLEROSUS is a benign vulvar dermatosis
- 6. **Thin snake skin**: lichen SCLEROSUS causes smooth white plaques on thin "parchment-like" tissue
- 7. **Thinned branch**: on histology, lichen SCLEROSUS appears as thinned epidermis overlying amorphous sclerotic stroma
- 8. Crab-patterned snake: lichen SCLEROSUS has a small risk of progression to vulvar squamous cell carcinoma
- 9. **Purple lichen at hole**: lichen PLANUS can present in the mouth or non-vulvar skin
- 10. **Spider web**: lichen PLANUS can appear as a fine net-like white plaques (Wickham striae) in the oral mucosa
- 11. **Purple polygonal lichen spots**: lichen PLANUS can appear as purple polygonal papules
- 12. Chronically scratching lichen : lichen SIMPLEX CHRONICUS presents with severe pruritus \rightarrow chronic scratching
- 13. **Leathery bark**: lichen SIMPLEX CHRONICUS appears as leathery white plaques with excoriations (due to chronic scratching)
- 14. **Thick flaking bark**: on histology, lichen simplex chronicus appears as epithelial thickening (due to squamous cell hyperplasia) with hyperkeratosis
- 15. "View INside": vulvar intraepithelial neoplasia (VIN) (a precursor to SCC) is analogous to cervical intraepithelial neoplasia (CIN)
- 16. **Pill bugs**: Vulvar dysplasia (VIN) is caused by high-risk HPV subtypes (e.g. 16, 18)
- 17. **ONE-THIRD dysplastic leaves**: VIN 1 is MILD dysplasia involving the lower ONE-THIRD of the epithelium

- 18. **TWO-THIRD dysplastic leaves**: VIN 2 is MODERATE dysplasia involving the lower TWO-THIRDs of the epithelium
- 19. **More than TWO-THIRD dysplastic leaves**: VIN 3 is SEVERE dysplasia involving MORE THAN TWO-THIRDS or full thickness of the epithelium
- 20. **Invading leaves**: VIN can progress to vulvar squamous cell carcinoma
- 21. **Pearl necklace**: vulvar squamous cell carcinoma has keratin pearls and intercellular bridges (similar to other squamous carcinomas)
- 22. **Striated leaves**: embryonal rhabdomyosarcoma is a skeletal muscle cancer of the vagina
- 23. **Young girl with cystic raspberries**: embryonal rhabdomyosarcoma occurs in young girls and presents as a jellylike cystic mass protruding from the vagina
- 24. **Translucent cellular glass**: clear cell adenocarcinoma (appears as clear vacuolated cells on histology)
- 25. **Columnar desk**: vaginal adenosis (exocervix and vagina develop columnar epithelium [rather than squamous])
- 26. "**DESk"**: women exposed to diethylstilbestrol (DES) in utero are at increased risk for clear cell adenocarcinoma and vaginal adenosis (and uterine and cervical anomalies)
- 27. "INF♀": DES is a synthetic estrogen
- 28. **Gardener in wolf shirt** : Gartner duct cysts are remnants of the Wolffian duct (mesonephric duct)
- 29. Cystic planter boxes on lateral wall: Gartner duct cysts appear on the lateral wall of the vagina

Reproductive & GU 2.1 - Reproductive Hormones Overview & Polycystic Ovary Syndrome (PCOS)





- 1. Flickering "GardeneR Hardware" sign : Gonadotropin Releasing Hormone (GnRH) (secreted by the hypothalamus in a PULSATILE fashion → travels to anterior pituitary via the hypophyseal portal system)
- 2. Anterior facing sac : anterior pituitary
- 3. Flower stimulating fertilizer (spilling out of pituitary sac): Follicle Stimulating Hormone (FSH) (pulsatile GnRH stimulates anterior pituitary → secretes FSH into systemic circulation)
- 4. **LigHt bulbs (spilling out of pituitary sac)**: Luteinizing Hormone (LH) (pulsatile GnRH stimulates anterior pituitary → secretes LH into systemic circulation)
- 5. Round pots : ovaries
- 6. "Thecal Matter": THECA cells of the ovarian follicle
- 7. "Light" shining on thecal matter : LH stimulates THECA cells to synthesize ANDROGENS
- 8. **Male trowel** : ANDROGENS (androstenedione, testosterone) are secreted by THECA cells (in response to stimulation by LH)
- 9. $\mbox{\bf Granular-looking soil}: \mbox{\bf GRANULOSA cells of the ovarian follicle}$
- 10. Flower Stimulator poured onto soil of Male Shovel : FSH stimulates GRANULOSA cells to convert ANDROGENS to ESTROGENS
- 11. Female flower: ESTROGENS (estrone, estradiol) are synthesized from androgens within GRANULOSA CELLS cells (in response to stimulation by FSH)
- 12. \$14 on Upper Row of pots : FOLLICULAR phase (lasts ~ 14 days) (begins on day 1 & ends with ovulation on day 14)
- 13. 0 cents on Lower Row of pots: LUTEAL phase (lasts ~ 14 days) (begins at ovulation on day 14 & ends before onset of menstruation on day 1)
- 14. **Growing estrogen flower buds**: in the FOLLICULAR phase, elevated FSH stimulates development of multiple follicles (which then produce more estrogen)
- 15. Trying to unplug "GardeneR Hardware" sign : NORMALLY, estrogen exerts NEGATIVE feedback on hypothalamus and pituitary
- 16. Inadvertently freeing Light bulbs: in FOLLICULAR phase, once ESTROGEN levels are sufficiently high, the pituitary turns on a POSITIVE feedback mechanism for ESTROGEN \rightarrow estrogen stimulates ENORMOUS release of LH from the pituitary ("LH surge")
- 17. **Big bright LigHt**: "LH surge" (toward end of FOLLICULAR phase) stimulates rupture of the dominant follicle and release of the secondary oocyte ("OVULATION")
- 18. **SINGLE large open flower** : during the FOLLICULAR phase, LH is responsible for developing a single dominant follicle → later ruptures during ovulation
- 19. **Yellow sunflower** : in the LUTEAL phase, the CORPUS LUTEUM ("yellow body") forms from the remnants of the ruptured ovarian follicle → secretes high levels of PROGESTERONE
- 20. **Uterine pot with proliferating plants**: in the LUTEAL phase, the uterine lining needs to be maintained in preparation for implantation of a fertilized egg
- 21. **PROGESTRO-dome**: in the LUTEAL phase, PROGESTERONE (secreted by the corpus luteum) maintains the uterine lining
- 22. Wilted yellow sunflower : the CORPUS LUTEUM regresses if the ovum is not fertilized within 14 days
- 23. Fallen PROGESTRO-dome : PROGESTERONE levels fall once the corpus luteum regresses)
- 24. Squirrel digging up uterine soil : PROGESTERONE withdrawal \to SLOUGHING off of Stratum Functionalis layer of the ENDOMETRIUM
- 25. **Bleeding from uterus pot**: sloughing of Stratum Functionalis of endometrium manifests as MENSES (ends LUTEAL phase)

- 26. Closed cystic buds in ovary pot: polycystic ovarian syndrome (PCOS) is the most common cause of ANOVULATION (lack of egg release during the menstrual cycle)
- 27. Male trowel in ovary pot : in PCOS, ovaries produce excessive amounts of ANDROGENS
- 28. Raising multiple male trowels: PCOS presents with hyperANDROGENISM (due to androgen production by the ovary)
- 29. **Bushy beard**: PCOS presents with HIRSUTISM (increased hair growth in mustache & beard area, chest, back, butt) (due to hyperANDROGENISM)
- 30. **Muscle-y arms**: PCOS presents with INCREASED MUSCLE MASS (due to hyperANDROGENISM)
- 31. **Balding head**: PCOS can present with male-pattern BALDNESS (due to hyperANDROGENISM)
- 32. Male trowel knocking down GRANULAR soil: excessive ANDROGENS in the ovaries cause GRANULOSA cell Dysfunction \rightarrow abnormal regulation of conversion of androgens to estrogen
- 33. Excessively large bud collection: in PCOS, ovaries contain collections of MULTIPLE unused FOLLICLES (excess androgens stimulate growth of excessive numbers of follicles)
- 34. Loudspeaker near large bud collection : on ULTRASOUND, PCOS displays enlarged ovaries with MULTIPLE FOLLICLES (appear similar to small cysts) in the periphery ("STRING OF PEARLS")]
- 35. **Empty pots**: PCOS presents with ANOVULATORY menstrual cycles (due to the effects of excessive androgens)
- 36. Irregular red drips from uterus can : PCOS presents with IRREGULAR MENSES (due to anovulatory cycles)
- 37. Barren tree: PCOS presents with INFERTILITY (due to anovulation)
- 38. "Welcome INSIDE" : PCOS often presents with insulin resistance and ELEVATED insulin levels
- 39. **Hanging candy pots**: PCOS is associated with metabolic syndrome and type 2 diabetes (due to insulin resistance)
- 40. Male trowel on "INSIDE" mat: HIGH INSULIN levels can EXACERBATE hyperANDROGENISM (insulin sensitizes androgen-producing theca cells to LH) (as seen in PCOS)
- 41. Large female stake in endometrial soil : in PCOS, the endometrium is exposed ONLY to ESTROGEN, NO PROGESTERONE (no ovulation \rightarrow no luteal phase \rightarrow no corpus luteum to secrete progesterone)
- 42. **Crab grass in uterine pot**: PCOS increases the risk of ENDOMETRIAL HYPERPLASIA & CANCER (endometrium exposed ONLY to estrogen without progesterone → estrogen releases persistent pro-growth signals → eventually abnormal cell growth)
- 43. **Thin woman**: in obese patients, WEIGHT LOSS is first line treatment for PCOS (decreased weight \rightarrow decreased insulin resistance \rightarrow reversed hyperinsulinism \rightarrow decreased androgen levels)
- 44. **Flourishing flower**: in obese patients, weight loss can start OVULATORY cycles and restore FERTILITY (weight loss \rightarrow \rightarrow decreased androgen levels \rightarrow normalized hormonal pathways)
- 45. **OCP headphones**: PCOS can be treated with combined estrogen-progestin oral contraceptives (OCPs) (VERY ELEVATED ESTROGEN suppresses LH secretion → DECREASED ANDROGEN production)
- 46. Normal endometrial plants: OCPs DECREASE the risk of HYPERPLASIA & CANCER (because PROGESTERONE stabilizes endometrial lining)
- 47. "Running is Life" METAPHOR: PCOS accompanied by insulin-resistance can be treated with METFORMIN (reversal of hyperinsulinism → decreased androgen levels and restored ovulation)

Reproductive & GU 2.2 - Amenorrhea



- 1. **Spotless white dress**: AMENORRHEA (absence of menses) can be PRIMARY [no history of menses] or SECONDARY [prior menses]
- 2. "Quinceanera": PRIMARY amenorrhea is absence of menses by age 15 (or 13 if NO secondary sex characteristics [breast development, pubic hair])
- 3. Baby tearing "Gardner's Hardware" bag : CONGENITAL GnRH DEFICIENCY (causes PRIMARY amenorrhea)
- 4. Falling FSH fertilizer & LH light : CONGENITAL GnRH DEFICIENCY leads to decreased secretion of FSH & LH by pituitary
- 5. Wilted female flowers: CONGENITAL GnRH DEFICIENCY leads to decreased secretion of ESTROGEN by ovaries (because of decreased FSH and LH)
- 6. Smiley shirt with "X"d breasts: CONGENITAL GnRH DEFICIENCY presents with ABSENCE of SECONDARY SEX characteristics (breast development, public hair) (due to total lack of estrogen)
- 7. Pot of wilting female flowers: OVARIAN dysfunction (can cause PRIMARY amenorrhea)
- 8. Fallen flowers with female symbols : PRIMARY amenorrhea due to OVARIAN dysfunction presents with LOW ESTROGEN
- 9. PRIMARY amenorrhea due to OVARIAN dysfunction presents with ELEVATED FSH & LH (due to decreased negative feedback on hypothalamus and pituitary):
- 10. **TURNING X-shaped pinwheel**: TURNER syndrome (45 XO genotype) (causes PRIMARY amenorrhea due to OVARIAN DYSFUNCTION)
- 11. Streaks of FIBROUS plant in wilting ovarian pot : in TURNER syndrome, ovarian follicles are replaced by FIBROUS connective tissue ("STREAK GONADS") → atrophic ovaries and no estrogen production
- 12. **Smiley shirt with "X"d breasts**: TURNER syndrome presents with ABSENCE of SECONDARY SEX characteristics (due to lack of estrogen)
- 13. **Mule attempting to be absent**: MULLERIAN AGENESIS (MRKH syndrome) (agenesis of FEMALE UPPER GENITAL TRACT) (causes PRIMARY amenorrhea)
- 14. Mule with floppy fallopian ears, uterine head, proximal vaginal tongue: MULLERIAN DUCTS normally develop into fallopian tubes, uterus, upper ½ of vagina
- 15. **NORMAL white dress**: MULLERIAN AGENESIS presents with PRIMARY amenorrhea (due to absence of uterus and proximal vagina) but NORMAL development of SECONDARY SEX characteristics (because ovaries are intact)
- 16. **Unable to break through IMPERFORATE bag**: IMPERFORATE HYMEN (failure of central hymen to degenerate during development → sheet of connective tissue OBSTRUCTING OUTFLOW from vagina) (causes PRIMARY amenorrhea)
- 17. **NORMAL white dress**: IMPERFORATE HYMEN presents with PRIMARY amenorrhea (due to obstruction of outflow from vagina) but NORMAL development of SECONDARY SEX characteristics (because ovaries are intact)
- 18. **Grimacing**: IMPERFORATE HYMEN presents with CYCLIC ABDOMINAL/PELVIC PAIN due to pooling of menstrual blood in uterus and vagina
- 19. **Pregnant woman**: PREGNANCY is the most common cause of SECONDARY amenorrhea
- 20. **Trampling "Gardner's Hardware"**: ACQUIRED GnRH DEFICIENCY (causeS SECONDARY amenorrhea)

- 21. Excessive running: ACQUIRED GnRH DEFICIENCY is caused by EXCESSIVE EXERCISE or severely LOW CALORIC INTAKE (anorexia nervosa) (insufficient calories to support pregnancy → hypothalamus decreases secretion of GnRH → amenorrhea)
- 22. Falling FSH fertilizer & LH light : ACQUIRED GnRH deficiency leads to DECREASED secretion of FSH & LH by pituitary
- 23. Wilted female flowers: ACQUIRED GnRH DEFICIENCY leads to DECREASED secretion of ESTROGEN by ovaries (due to decreased FSH and LH)
- 24. "PL" baby bottle: HYPERPROLACTINEMIA can cause SECONDARY amenorrhea (because prolactin inhibits secretion of GnRH by the hypothalamus)
- 25. **Enlarged pituitary bottle bag** : PROLACTINOMAS (benign tumors that secrete prolactin) are the most common cause of HYPERprolactinemia (→ SECONDARY amenorrhea)
- 26. Milk spraying from PL bottle: PROLACTINOMAS can cause GALACTORRHEA (lactation) (due to elevated prolactin levels)
- 27. **Broken double ropes**: antipsychotic medications with DOPAMINE (D2) RECEPTOR ANTAGONIST effects can cause HYPERprolactinemia (by inhibiting inhibition of prolactin secretion by pituitary) (→ SECONDARY amenorrhea)
- 28. **Torn "HYPE" and beat-up PITUITARY punching bag**: HYPOTHALAMIC and PITUITARY dysfunction can cause SECONDARY amenorrhea
- 29. Removing ovarian pot: SURGICAL REMOVAL OF OVARIES causes SECONDARY amenorrhea
- 30. Blasting LH light & deploying FSH fertilizer: SECONDARY amenorrhea due to OVARIAN REMOVAL presents with ELEVATED FSH & LH (due to decreased negative feedback on hypothalamus and pituitary)
- 31. "PAUSE" sign on "12 months" video : menoPAUSE (SECONDARY amenorrhea lasting > 12 months in the absence of any other pathology)
- 32. **Dwindling follicular flowers**: physiologic MENOPAUSE is caused by depletion of ovarian follicles (→ decreased estrogen → lack of feedback inhibition of anterior pituitary → INCREASED FSH specifically)
- 33. Older lady sweating: menopausal transition (PERIMENOPAUSE) can present with HOT FLASHES and NIGHT SWEATS
- 34. Older lady with desert skirt : menopausal transition (PERIMENOPAUSE) can present with VAGINAL DRYNESS
- 35. Cracked boney table: MENOPAUSE leads to DECREASED BONE DENSITY (estrogen no longer available to inhibit resorption of bone by osteoclasts)
- 36. Younger woman sweating in desert skirt: PRIMARY OVARIAN INSUFFICIENCY (signs of menopause before age 40) (presents with irregular menses, menopausal symptoms, high FSH levels)
- 37. **ASH on collapsed uterus cake**: ASHERMAN syndrome (SCARRING OF ENDOMETRIUM preventing normal buildup and shedding of endometrial lining) (can cause SECONDARY amenorrhea)
- 38. Bacterial lanterns around uterus cake: repeated ENDOMETRIAL INFECTION can cause ASHERMAN syndrome → SECONDARY amenorrhea
- 39. Knives near uterus cake : UTERINE SURGERY can cause ASHERMAN syndrome \rightarrow SECONDARY amenorrhea

Reproductive & GU 2.3 - Ovarian Cysts & Epithelial Ovarian Cancer



- 1. **Cystic balloons**: follicular (physiologic) cysts occur if a mature follicle does not rupture and continues to grow
- 2. **Bursting cystic balloon**: follicular cysts can rupture, causing sterile peritonitis and severe abdominal pain
- 3. **Torsing cystic balloon**: follicular cysts can cause ovarian torsion and severe abdominal pain
- 4. **Yellow luteal flower**: corpus luteum cysts occur if the corpus luteum (the yellow body left behind after egg rupture) fills with fluid
- 5. **Pregnant woman tending to luteal flower**: corpus luteum cysts often occur in pregnancy
- 6. Palpating the ovary basket: all types of ovarian tumors (and benign cysts!) can present with a palpable adnexal mass
- 7. Enlarged egg: ovarian tumors (EPITHELIAL (most common), germ cell, and sex-cord stromal tumors)
- 8. **Cuboidal ovarian rim**: ovarian surface epithelium is composed of cuboidal cells
- 9. **Broccoli**: mutations in BRCA1 and BRCA2 (tumor suppressor genes) increase the risk of EPITHELIAL ovarian cancer and breast cancer
- L-inch worm: Lynch syndrome (hereditary nonpolyposis colon cancer - HNPCC) increases the risk of EPITHELIAL ovarian, colon, and endometrial cancer
- 11. **Torn basket rim**: repeated damage to the ovarian surface epithelium increases the risk of EPITHELIAL ovarian tumor
- 12. **Eggs spilling out**: increased ovulation increases the risk of EPITHELIAL ovarian cancer (increased damage to surface epithelium)
- 13. **Empty eggs**: nulliparity increases the risk of EPITHELIAL ovarian cancer (due to increased lifetime ovulatory cycles)
- 14. "Early bloomer": early menarche increases the risk of EPITHELIAL ovarian cancer (due to increased lifetime ovulatory cycles)
- 15. ["Late deciduous": late menopause increases the risk of EPITHELIAL ovarian cancer (due to increased lifetime ovulatory cycles)
- 16. **Endometrial soil in egg**: endometriosis increases the risk of EPITHELIAL ovarian cancer (due to oxidative stress and inflammation)
- 17. **Locked OCP basket**: oral contraceptive pills (OCPs) decrease the risk of EPITHELIAL ovarian cancer (due to decreased number of lifetime ovulatory cycles)
- 18. **Dilated inner tube**: EPITHELIAL ovarian cancer can present with ascites (due to peritoneal seeding)
- 19. **Obstructing intestinal pool**: EPITHELIAL ovarian cancer can present with bowel obstruction (due to seeding on the bowel)

- 20. **Wet pleural shirt**: EPITHELIAL ovarian cancer can present with malignant pleural effusions (due to pleural metastasis)
- 21. **CA-125** : CA-125 is the tumor marker for EPITHELIAL ovarian cancer (used to track response to therapy)
- 22. **Bilateral wet baskets**: SEROUS tumors are the most common EPITHELIAL ovarian tumor (commonly bilateral, may be benign [cystadenoma] or malignant [cystadenocarcinoma])]
- 23. Columnar picket fence with ciliary grass: on histology, SEROUS tumors have fallopian tube-like ciliated columnar cells
- 24. **Concentrically laminated roses**: on histology, SEROUS tumors have psammoma bodies (concentrically laminated, calcified spheric deposits)
- 25. Calcified bone buried at the roses : psammoma bodies are calcified
- 26. **Mucusy eggs**: MUCINOUS tumors are a type of EPITHELIAL ovarian cancer (may be benign [cystadenoma] or malignant [cystadenocarcinoma])
- 27. **Pink mucus eggs**: on histology, MUCINOUS tumors are lined by light-pink mucin-secreting epithelial cells
- 28. **Mucusy abdomen**: MUCINOUS tumors can cause pseudomyxoma peritonei (peritoneal cavity filled with jelly-like mucin)
- 29. $\mbox{\bf BRown hen}$: BRENNER tumors are a type of EPITHELIAL ovarian tumor (usually benign)
- 30. **Nest with transitioning chick**: on histology, BRENNER tumors contain nests of transitional-type epithelium (similar to urinary tract)
- 31. **Coffee-bean eggs**: on histology, BRENNER tumors contain coffee bean nuclei
- 32. **Endometrial crabgrass**: ENDOMETRIOID ovarian cancer is a type of EPITHELIAL ovarian cancer (malignant, may present with coexisting endometrial carcinoma)
- 33. **Purple glandular grass**: on histology, ENDOMETRIOID ovarian cancer appears similar to endometrial carcinoma (glands with many purple nuclei)
- 34. Cooking burgers with crab apron: KRUKENBERG tumors are gastric cancer metastatic to the ovaries
- 35. **Burger flipped into bilateral baskets**: KRUKENBERG tumors often occur bilaterally
- 36. **Signet rings**: on histology, KRUKENBERG tumors have signet ring cells (mucin displacing nucleus against cell membrane)

Reproductive & GU 2.4 - Ovarian Neoplasms (Germ Cell & Sex Chord-Stromal Tumors)





- 1. Palpating the ovary basket : all types of ovarian tumors (and benign cysts!) can present with a palpable adnexal mass
- GERMinating seeds : ovarian GERM CELL tumors derive from primordial germ cells and include cystic teratomas, dysgerminomas, and yolk sac tumors
- 3. Three-layered terrarium: CYSTIC TERATOMAS are usually benign, contain cells from three germ layers (ectoderm, mesoderm, and endoderm), and occur in ages 10-30 years (GERM CELL TUMOR)
- 4. Hair grass, glandular tissue, and teeth rocks: the most prominent component is ectodermal tissue (hair, sebaceous glands, and teeth)(in MATURE CYSTIC TERATOMAS)
- 5. **Bright spot**: calcification from teeth and bone causes bright spots on imaging (in CYSTIC TERATOMAS)
- 6. Twisting terrarium : CYSTIC TERATOMAS can present with ovarian torsion \rightarrow severe abdominal pain
- 7. **"Shrooma" ovarii terrarium**: STRUMA OVARII (a cystic teratoma subtype) contain functioning thyroid tissue (GERM CELL TUMOR)
- 8. **Hyperthyroid bow tie**: STRUMA OVARII tumors secrete thyroid hormone → hyperthyroidism
- 9. **Shroom follicles**: on histology, STRUMA OVARII have thyroid follicles filled with eosinophilic material
- 10. **Glowing shrooms**: STRUMA OVARII demonstrate uptake of radioactive iodine-123
- 11. Crab terrarium with undifferentiated rocks: IMMATURE TERATOMAS are malignant and have poorly differentiated tissues (GERM CELL TUMOR)
- 12. **DYS-GERMINATOR dishwasher**: DYSGERMINOMAS are malignant and occur in adolescents (GERM CELL TUMOR)
- 13. **Sea-man superhero**: DYSGERMINOMAS are the counterpart of testicular seminomas in males (similar histology)
- 14. **B-hCG babysitter tending yellow flower baby**: B-hCG is a tumor marker for DYSGERMINOMAS
- 15. **Dehydrated LDH milk**: lactate dehydrogenase (LDH) is a tumor marker for DYSGERMINOMAS
- 16. "Fried eggs": in DYSGERMINOMAS, histology shows "fried egg cells" (large central nuclei with clear cytoplasm)
- 17. ${f Egg\ yolk}$: YOLK SAC (endodermal sinus) tumors are usually malignant and occur in girls and young women (GERM cell tumors)
- 18. "Alf's Fresh Produce" : α -fetoprotein (AFP) is a tumor marker for YOLK SAC tumors
- 19. **Egg chiller**: in YOLK SAC tumors, histology shows Schiller-Duval bodies (papillary structure with central vessel)
- 20. **Egg spots with red center** : Schiller-Duval bodies are papillary structure with a central vessel

- 21. **Decorative cords**: ovarian SEX CORD-STROMAL TUMORS include thecoma-fibromas, granulosa-theca tumors, and sertoli-leydig tumors
- 22. **COMBing through hair FIBERS**: THECOMA-FIBROMAS are usually benign and present in perimenopausal women (SEX-CORD STROMAL TUMORS)
- 23. "MY EGGS": THECOMA-FIBROMAS cause Meigs syndrome (ascites, pleural effusion)
- 24. **Dilated inner tube**: Meigs syndrome causes ascites (THECOMA-FIBROMA)
- 25. **Wet pleural shirt**: Meigs syndrome causes pleural effusion (THECOMA-FIBROMA)
- 26. **Yellow eggs**: THECOMA-FIBROMAS appear yellow on gross pathology due to theca cells
- 27. **GRANNY**: GRANULOSA-THECA tumors are low-grade malignant and can occur at any age (but usually in perimenopause) (SEX-CORD STROMAL tumor)
- 28. **Estrogen earrings**: GRANULOSA-THECA tumors produce estrogen (causes precocious puberty, bleeding, endometrial hyperplasia/cancer)
- 29. Young girl with makeup : GRANULOSA-THECA tumors can cause precocious puberty due to estrogen production
- 30. **Spots of red dye on granny**: GRANULOSA-THECA tumors can cause abnormal uterine bleeding or postmenopausal bleeding due to estrogen production
- 31. **Granny INHIBITING inappropriate behavior**: INHIBIN is the tumor marker for GRANULOSA-THECA tumors
- 32. CALLing Exner: on histology, GRANULOSA-THECA tumors have call-Exner bodies (cuboidal granulosa cells in rosette pattern with eosinophilic centers)
- 33. **Pink rotary dial with square holes**: Call-Exner bodies consist of cuboidal granulosa tumors in rosette pattern with eosinophilic center
- 34. ${f Coffee\ cup}$: cells in Call-Exner bodies have "coffee bean" nuclei (in GRANULOSA-THECA tumors)
- 35. **Yellow chick**: GRANULOSA-THECA tumors appear yellow on gross pathology due to lipid in theca cells
- 36. **Ladybugs**: SERTOLI-LEYDIG tumors can be benign or malignant and occur at any age (but primarily in young adults)(SEX-CORD STROMAL tumor)
- 37. Androgen egg holder: SERTOLI-LEYDIG tumors produce androgens (causing hirsutism, clitoromegaly, voice changes)
- 38. **Egg dye mustache**: SERTOLI-LEYDIG tumors can cause hirsutism due to production of androgens
- 39. **TUBULAR egg holders**: on histology, SERTOLI-LEYDIG tumors appear as tubular structures lined by round Sertoli cells

Reproductive & GU 3.1 - Hydatidiform Mole & Choriocarcinoma



- Villous-shaped TROPHY case: the TROPHOBLAST forms the chorionic villi (part of placenta that derives from the embryo) (transfers oxygen and nutrients between mother and fetus) (composed of two layers [SYNCYTIOtrophoblast & CYTOtrophoblast])
- 2. SYNCHRONIZED swimming trophies on outer shelf: the SYNCYTIOtrophoblast is the outer layer of villi (comes into direct contact with maternal blood) (composed of large cells with many nuclei) (secretes β-hCG and human placental lactogen [HPL])
- 3. CYTO-CYCLING trophies on inner shelves: the CYTOtrophoblast is the inner layer of villi (composed of many mononuclear cells)
- 4. **Prominent stressed** β -hCG babysitter: β -human chorionic gonadotropin (β -hCG) is EXTREMELY elevated in many gestational trophoblastic diseases (because β -hCG is secreted by trophoblast [specifically SYNCYTIOtrophoblast])
- Stuffed mole: Hydatidiform MOLE (alternatively "Molar Pregnancy" because they
 are a type of abnormal pregnancy) (most common gestational trophoblastic
 disease) (can be Complete or Partial)
- 6. Young mom & older mom: extremes of maternal age (<15 or >35) increase the risk of molar pregnancy
- Repeated mole: history of molar pregnancy increases the risk of another molar pregnancy)
- Overturned baby carriage: history of miscarriage increases the risk of molar pregnancy
- 9. Shirt COMPLETELY stuffed with mole : COMPLETE hydatidiform mole (causes EXTREMELY high $\beta\text{-hCG}$ levels)
- 10. Round mirror : EMPTY ovum
- 11. Nerf ball with "X" fin: NORMAL sperm (haploid 23X)
- 12. "X" nerf ball duplicated in empty mirror : most COMPLETE moles are 46XX (EMPTY ovum is fertilized by one 23X sperm \rightarrow sperm DUPLICATES within egg \rightarrow 46XX zygote)
- 13. Uterus vase completely filled with marbles: in a COMPLETE mole, all chorionic villi are COMPLETELY ABNORMAL (large, disordered, and edematous "hydropic"]) ("bunch of grapes")
- 14. **Uterus vase dripping red** : COMPLETE moles present with vaginal bleeding in early pregnancy
- 15. Oversized pregnant belly: COMPLETE moles present with enormous uterus (ENLARGED out of proportion to gestational age
- 16. Bullhorn: ultrasound
- 17. **Detecting feather storm**: on ultrasound, COMPLETE moles display a "snowstorm" pattern (diffuse echogenic material in uterus)
- 18. Green face : COMPLETE moles can present with hyperemesis gravidarum (severe nausea & vomiting) (due to EXTREMELY high β -hCG levels)
- 19. Large bowtie: COMPLETE moles can present with HYPERthyroidism (due to EXTREMELY high β -hCG levels [hCG shares common alpha subunit with TSH \rightarrow stimulates TSH receptors in thyroid])

- 20. **Bilateral cystic flowers** : COMPLETE moles can present with bilateral thecalutein cysts (multilocular ovarian cysts) (due to EXTREMELY high β-hCG levels)
- 21. Young child with steaming hair CLAMP: COMPLETE moles can present with early onset preeclampsia (at <20 weeks gestational age) (due to abnormal placentation)
- 22. Crab painting near complete mole: hydatidiform moles can undergo malignant transformation into choriocarcinoma (or other gestational trophoblastic neoplasm) (greater risk in COMPLETE > PARTIAL) (suspect if β -hCG levels don't decrease after pregnancy termination)
- 23. Shirt PARTIALLY stuffed with mole : PARTIAL hydatidiform mole (causes SLIGHTLY high β -hCG levels
- 24. Labeled laundry basket: PARTIAL moles are 69 XXX, XXY, or XYY (TRIPLOID and contain at least one X chromosome)
- 25. Two "X" nerf balls entering FULL basket : PARTIAL moles are TRIPLOID (NORMAL ovum is fertilized by TWO normal sperm → triploid zygote)
- 26. **Uterus vase with baby doll & marbles** : a PARTIAL mole contains fetal PARTS (in addition to abnormal villi)
- 27. **Normal pregnant belly**: PARTIAL moles present with uterine size that is NORMAL or slightly small for gestational age
- 28. **Uterus vase dripping red**: COMPLETE moles present with vaginal bleeding in early pregnancy
- 29. **"57" on term paper**: PARTIAL moles are POSITIVE for p57 (protein expressed by a MATERNAL allele) (in contrast, COMPLETE poles are NOT positive because of absence of maternal DNA)
- 30. **CHOIR music box**: CHORIOCARCINOMA (most common gestational trophoblastic neoplasm) (malignant tumor arising from trophoblast) (occurs after pregnancy)
- 31. Crabs scattered outside uterus box : CHORIOCARCINOMAS are usually metastatic at time of diagnosis
- 32. β-hCG babysitter with kid, empty carriage, baby doll keychain, stuffed mole : CHORIOCARCINOMAS can occur after ANY pregnancy type (normal, preterm, terminated, ectopic, or molar) (but most common after COMPLETE mole)
- 33. **Disordered TROPHIES without marbles**: on histology, CHORIOCARCINOMA displays abnormal proliferation of CYTOtrophoblast & SYNCYTIOtrophoblast cells (but NO chorionic villi)
- 34. **Dripping blood from uterus box**: CHORIOCARCINOMAS can present with vaginal bleeding
- 35. β -hCG babysitter : CHORIOCARCINOMAS present with rapidly rising β -hCG levels
- 36. Crab pin on shirt: CHORIOCARCINOMAS commonly metastasize to the lungs (often prior to time of diagnosis)
- 37. **Bleeding from crab pin**: CHORIOCARCINOMAS can present with hemoptysis (due to lung metastasis)
- 38. **Methotrexate chopsticks**: CHORIOCARCINOMA is treated with methotrexate (highly effective)

Reproductive & GU 3.2 - Gestational Disorders





- Baby keychain outside uterus purse : ECTOPIC PREGNANCY (embryo implants outside uterus)
- 2. Two straps connecting uterus purse to ovarian buttons: ECTOPIC pregnancy usually implants in the FALLOPIAN tubes (may also occur in ovaries or abdominal cavity)
- 3. **Key chain at widened mid-strap**: the AMPULLA of the fallopian tube is the most common site of ECTOPIC pregnancy (same location as fertilization)
- 4. **Knot in curtains**: tubal ligation increases the risk of ECTOPIC pregnancy
- 5. "DESk": exposure to diethylstilbestrol (DES) (as a fetus) increases the risk of ECTOPIC pregnancy (due to abnormal fallopian tube anatomy)
- Fallopian chandelier with fiery candles: pelvic inflammatory disease (PID) increases the risk of ECTOPIC pregnancy (due to fallopian tube injury)
- 7. **Soil spilling out of uterus planter**: ENDOMETRIOSIS increases the risk of ECTOPIC pregnancy (due to fallopian tube injury)
- 8. **Poking belly with name tag**: ECTOPIC pregnancy presents with acute lower abdominal pain
- 9. **Bleeding uterus pot**: ECTOPIC pregnancy presents with vaginal bleeding
- Deciduous plants in uterus pot: in ECTOPIC pregnancy, a sample of uterine lining displays DECIDUALIZED endometrium (thickened endometrium, normally occurs in pregnancy)
- 11. **Torn curtain**: untreated ECTOPIC pregnancy can lead to fallopian tube rupture
- 12. **Passed out from bloody stomach**: untreated ECTOPIC pregnancy can lead to life-threatening intra-abdominal hemorrhage (due to ruptured fallopian tube)
- 13. Meat sticks: ECTOPIC pregnancy can be treated with methotrexate
- 14. Baby holding HPL formula: the SYNCYTIOtrophoblast (originates from fetal tissue) secretes human placental lactogen (HPL)
- Pregnant woman with HPL & candy jar: GESTATIONAL DIABETES (new onset diabetes during pregnancy, resolves after delivery) (caused by increased HPL)
- 16. **Resistance from welcome "INSIDE" mat**: HPL increases INSULIN RESISTANCE in mother
- 17. **Pregnant woman with raised candy jar**: GESTATIONAL DIABETES presents with hyperglycemia (HPL → insulin resistance → hyperglycemia IF pancreas is unable to compensate by increasing insulin secretion)

- 18. **Drinking sugary party punch**: pregnant women are screened for GESTATIONAL DIABETES with an oral glucose tolerance test (drink glucose solution → measure serum glucose levels at specified times)
- 19. **Smiling mother**: GESTATIONAL DIABETES resolves once baby is delivered (delivery of placenta → HPL levels decrease to pre-pregnancy levels)
- 20. **Dia-sweeties candy**: GESTATIONAL DIABETES is associated with a higher risk of developing DM2 later in life
- 21. **Pink plate implanted over trash opening**: PLACENTA PREVIA (implantation of placenta over cervical OS [opening of cervix] (causes painLESS vaginal bleeding DURING pregnancy)
- 22. **Spilling red food**: PLACENTA PREVIA presents with vaginal bleeding during pregnancy (placenta implanted over cervical os → disruption of placental attachment as cervix changes size and shape in late pregnancy → bleeding)
- 23. Happy face: in PLACENTA PREVIA, vaginal bleeding is PAINLESS
- 24. **Shirt cut by surgical knife**: previous Cesarean delivery increases the risk of PLACENTA PREVIA
- 25. Twins: multiple gestation increases the risk of PLACENTA PREVIA
- 26. **ABRUPTLY dropping pink plate**: PLACENTAL ABRUPTION (premature detachment of placenta from uterine wall) (causes PAINFUL vaginal bleeding DURING pregnancy)
- 27. **Grimacing**: in PLACENTAL ABRUPTION, vaginal bleeding is PAINFUL
- 28. **Concealed plate**: PLACENTA ABRUPTION sometimes presents WITHOUT vaginal bleeding ("CONCEALED abruption") (bleeding DOES occur, but blood is trapped between fetal membranes and uterine wall)
- 29. **Cast**: physical trauma during pregnancy increases the risk of PLACENTAL ABRUPTION
- 30. **Steaming angry**: hypertension increases the risk of PLACENTAL ABRUPTION
- 31. **Hot "Coca"**: cocaine use increases the risk of PLACENTAL ABRUPTION
- 32. Cigarette: smoking increases the risk of PLACENTAL ABRUPTION
- 33. **Shaking belly**: PLACENTAL ABRUPTION presents with painful prolonged uterine contractions
- 34. Red food clump on back of plate: PLACENTAL ABRUPTION may display a RETROPLACENTAL HEMATOMA (clot behind placenta) on ultrasound

Reproductive & GU 3.2 - Gestational Disorders





- 35. **Mother with baby and floppy uterus bag**: UTERINE ATONY (uterus fails to contract after delivery) (most common cause of postpartum hemorrhage [PPH])
- 36. Floppy uterus spilling red clothes : in UTERINE ATONY, uterus fails to contract after placenta detaches from uterine wall → decreased mechanical compression of exposed uterine vessels → hemorrhage
- 37. Falling 8 ball: COAGULOPATHIES can cause PPH hemorrhage (von Willebrand disease, factor VIII deficiency)
- 38. Torn pants: LACERATIONS can cause PPH
- 39. Pink plate adhered to smooth muscle tile: PLACENTA ACCRETA (a type of morbidly adherent placenta) (placenta attaches directly to uterine muscle [myometrium] rather than to endometrium [decidua]) (causes PPH)
- 40. **Plate WITHIN smooth muscle tile**: PLACENTA INcreta (a type of morbidly adherent placenta) (placenta invades INto myometrium) (causes PPH)
- 41. **Plate PAST smooth muscle tile**: PLACENTA PERcreta (a type of morbidly adherent placenta) (placenta invades PAST myometrium → into serosa of uterine wall or surrounding organs) (causes PPH)
- 42. **Bleeding pink plate**: a morbidly adherent placenta (ACCRETA, INcreta, PERcreta) causes severe PPH when delivery of placenta is attempted (because of direct attachment to myometrium)
- 43. **DICe**: severe hemorrhage DURING pregnancy or AFTER delivery (PPH) can lead to disseminated intravascular coagulation (DIC)
- 44. **Steaming red pipes**: HTN during pregnancy may be due to PRE-EXISTING hypertension (onset before pregnancy or BEFORE 20 wks gestation) or HYPERTENSIVE DISORDERS OF PREGNANCY (onset > 20 wks) (gestational HTN, preeclampsia, eclampsia, HELLP syndrome)
- 45. **Class of "20"**: Gestational HTN (ISOLATED high BP with onset AFTER 20 wks gestation) (treated with hydralazine, alpha-methyldopa, labetalol)
- 46. **Steaming hair CLAMP**: PREECLAMPSIA (new onset HTN after 20 wks gestation PLUS proteinuria OR signs of end-organ dysfunction)
- 47. **Proteinaceous hair**: PREECLAMPSIA frequently presents with proteinuria (systemic endothelial dysfunction → injury to renal capillary endothelium → increased permeability of glomerulus
- 48. **Clutching head**: PREECLAMPSIA can present with headache (sign of end-organ dysfunction)
- 49. **Blurry reflection**: PREECLAMPSIA can present with vision changes (sign of end-organ dysfunction)

- 50. 50Plug attached abnormally to placental outlet: PREECLAMPSIA is caused by abnormal development of blood vessels to the placenta ("abnormal placentation")
- 51. Fiery burned placental outlet: in PREECLAMPSIA, abnormal blood flow to placenta leads to placental hypoxia, inflammation, and infarction → release of anti-angiogenic and inflammatory factors into circulation → systemic endothelial dysfunction
- 52. **Sleeve constricted by red cord**: in PREECLAMPSIA, HTN is caused by systemic VASOCONSTRICTION (systemic endothelial dysfunction → decreased production of vasodilators [prostaglandin E2, prostacyclin] AND increased production of vasoconstrictors [thromboxane A2])
- 53. **Poofy pants**: PREECLAMPSIA can present with peripheral edema (systemic endothelial dysfunction → increased capillary permeability) (also may be secondary to severe proteinuria)
- 54. Large liver bag : PREECLAMPSIA leads to liver swelling (--> stretching of liver capsule \rightarrow RUQ pain)
- 55. Large liver bag : PREECLAMPSIA leads to liver swelling (--> stretching of liver capsule \rightarrow RUQ pain)
- 56. **Raised candy jar**: pre-existing diabetes increases the risk of PREECLAMPSIA (due to vascular insufficiency)
- 57. **Worn kidney purse**: chronic kidney disease increases the risk of PREECLAMPSIA (due to vascular insufficiency)
- 58. **Twins**: multiple gestation increases the risk of PREECLAMPSIA (due to increased placental size without corresponding increase in blood flow)
- 59. Grey hair: older age is a risk factor for PREECLAMPSIA
- 60. **Holding up "1" finger** : first pregnancy is a risk factor for PREECLAMPSIA
- 61. **Shaking from hair CLAMP complication**: ECLAMPSIA (generalized tonic-clonic seizures caused by severe PREeclampsia)
- 62. "**HELLP**": HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelets)
- 63. Broken white plates: HELLP presents with low platelets
- 64. Falling red plate & schistocyte squashed tomatoes: HELLP presents with microangiopathic hemolytic anemia (displays schistocytes on peripheral smear)
- 65. ALT & AST tins falling from liver purse: HELLP presents with elevated transaminases (ALT & AST) (due to hepatic inflammation)
- 66. **MAGazine**: magnesium sulfate is administered to patients with PREECLAMPSIA in order to prevent seizures (ECLAMPSIA)



- Wet pregnant belly: POLYhydramnios (excessive amniotic fluid) (due to decreased swallowing of amniotic fluid by fetus OR increased fetal urination
- 2. **LARGE uterus box**: POLYhydramnios causes uterus to be LARGER than expected for gestational age
- Falling baby bottle full of unswallowed milk: POLYhydramnios can be caused by decreased swallowing of amniotic fluid (due to GI obstruction OR defect in swallowing center in the brain)
- 4. Tied-off duodenal diaper bag : DUODENAL ATRESIA can cause POLYhydramnios (GI obstruction → decreased swallowing of amniotic fluid
- Esophageal tube attached to tracheal slide : TRACHEOESOPHAGEAL FISTULA can cause POLYhydramnios (GI obstruction → decreased swallowing of amniotic fluid)
- Severed doll head : ANENCEPHALY (a neural tube defect) can cause POLYhydramnios (defect of swallowing center in the brain → decreased swallowing of amniotic fluid)
- 7. **Spilling large volume yellow juice**: POLYhydramnios can be caused by increased fetal urination (due to fetal high-output state OR maternal hyperglycemia)
- 8. Red confetti exploding from heart popper : POLYhydramnios can be caused by high fetal cardiac output
- 9. **Pale porcelain doll** : fetal ANEMIA can cause POLYhydramnios (insufficient RBCs \rightarrow increased cardiac output \rightarrow increased blood volume flowing through fetal kidneys \rightarrow increased urination)
- Throwing up candy: MATERNAL DIABETES can cause POLYhydramnios (excessive maternal glucose crosses placenta → fetal hyperglycemia → fetal polyuria)
- 11. Wrapped without intervening fluid layer: OLIGOhydramnios (insufficient amniotic fluid volume)
- 12. **SMALL uterus purse**: OLIGOhydramnios causes uterus to be SMALLER than expected gestational age
- 13. **Minimal yellow juice leaking from spilled cup**: OLIGOhydramnios is caused by decreased fetal urination (due to decreased production by kidneys OR lower urinary tract obstruction)
- 14. **Two popped kidney balloons**: BILATERAL RENAL AGENESIS can cause OLIGOhydramnios (due to insufficient urine production)

- 15. **Kidney composed of cystic balloons**: Autosomal Recessive POLYCYSTIC KIDNEY DISEASE can cause OLIGOhydramnios (due to insufficient urine production)
- 16. Exposing ACE card: exposure to ACE inhibitors or ARBs in utero can cause OLIGOhydramnios (because of decreased angiotensin II)
- 17. Floppy red angiotensin suspenders : exposure to ACE inhibitors or ARBs leads to DECREASED ANGIOTENSIN II → abnormal fetal kidney growth and development → insufficient urine production → OLIGOhydramnios
- 18. **Ribbons obstructing urethral streamer**: POSTERIOR URETHRAL VALVES can cause OLIGOhydramnios (obstruction at the proximal urethra [near bladder outlet] → decreased urination) (occurs only in males)
- 19. **Tightly wrapped Potter bear**: OLIGOhydramnios prevents the normal development, movement, and cushioning of the fetus \rightarrow leads to Potter sequence
- 20. **Twisted feet**: Potter sequence presents with clubbed feet (due to insufficient cushioning of fetus)
- 21. **Tiny lung lapels**: Potter sequence leads to pulmonary hypoplasia (due to insufficient amniotic fluid to swallow)



- Lobular pool : BREAST LOBULE (an EPITHELIAL component of breast) (location of milk production)
- Slip 'n slide: LACTIFEROUS DUCTS (an EPITHELIAL component of breast) (transports milk to the nipple)
- 3. **Slide exit**: NIPPLE (an EPITHELIAL component of breast) (releases breastmilk)
- 4. **Patio outside slide**: STROMA component of breast (fatty and fibrous connective tissue) (majority of breast volume in non-lactating state)
- 5. Flaming slide duct: MASTITIS (inflammation of breast ducts) (can occur any time, but most commonly caused by breastfeeding)
- 6. Red bathing suit: MASTITIS presents with erythema
- 7. Pained face: MASTITIS presents with breast pain
- 8. **Cradling baby**: breastfeeding is the most common cause of MASTITIS ("lactational mastitis")
- Kicking bikini : nipple trauma is the most common inciting event for development of MASTITIS
- 10. **Pooling water**: a GALACTOCELE (milk cyst) can form during lactation (nipple trauma from baby → poor milk drainage → duct obstruction → breast milk trapped within ducts → milk cyst
- 11. **Bikini turning red**: lactational MASTITIS occurs when bacteria proliferate in a galactocele (breast milk contains bacteria)
- 12. Golden staff: MASTITIS is most commonly caused by Staph aureus
- 13. **Steady water stream**: continuation of breastfeeding is an important part of treatment for lactational MASTITIS → relieves breast obstruction
- 14. **Thick white water**: MASTITIS may present with purulent nipple discharge
- 15. **Obstructed slide duct**: MAMMARY DUCT ECTASIA (lactiferous ducts are blocked by debris)
- 16. Flaming slide duct: in MAMMARY DUCT ECTASIA, duct obstruction leads to inflammation
- 17. **Grimacing**: MAMMARY DUCT ECTASIA presents with breast pain
- 18. **Dilated slide walls**: in MAMMARY DUCT ECTASIA, ducts are dilated ("ectasia")
- 19. **Leaking green water**: MAMMARY DUCT ECTASIA can present with thick ("inspissated") green nipple discharge
- White flowers on swimsuit: MAMMARY DUCT ECTASIA may display radio-opacities on mammogram (due to calcification of inspissated secretions)

- 21. Rotund kid wearing skull shirt outside slide: FAT NECROSIS (benign necrosis of breast adipose tissue [part of the STROMA])
- 22. **Cast**: FAT NECROSIS is most commonly caused by breast trauma ("traumatic fat necrosis")
- 23. **Grimacing**: FAT NECROSIS presents with breast pain
- 24. Irregular rocks: FAT NECROSIS presents with irregular breast mass
- 25. **Small white flowers**: FAT NECROSIS may display radio-opacities on mammogram (due to calcification of necrotic fat) (may be confused with breast cancer)
- 26. **Fibrous bush area**: FIBROCYSTIC CHANGE (may be NONproliferative or PROLIFERATIVE) (most common breast abnormality in premenopausal women)
- 27. **Small diffuse water balloons**: BREAST CYSTS (most common NON-proliferative lesion) (small cysts diffusely distributed throughout both breasts)
- 28. **Fibrous bush**: FIBROUS STROMAL proliferation (often co-occurs with breast cysts) → dense "lumpy" breast (a NON-proliferative lesion)
- 29. **Blue balloons**: BREAST CYSTS appear blue on gross pathology ("blue dome cysts")
- 30. **Small white flowers**: BREAST CYSTS can display radio-opacities on mammogram (due to calcification of fluid within cysts)
- 31. **Imitating an APE**: PAPILLARY APOCRINE change (growth of ductal epithelial cells that appear similar to apocrine glands) (a NON-proliferative lesion)
- 32. **Swim trunks with GRANULAR purple dots**: in PAPILLARY APOCRINE change, abnormal ductal epithelial cells appear GRANULAR (similar to apocrine glands) (but are NOT apocrine glands)
- 33. **Hit in the breast by non-proliferative balloon**: NONproliferative FIBROCYSTIC CHANGE presents with cyclic mastalgia (breast pain) in premenopausal women



- 34. **Benign bubbles filling pool**: EPITHELIAL HYPERPLASIA (proliferation of epithelial cells → eventually fills lumen of mammary ducts or lobules) (organized cells with small purple nuclei) (a BENIGN PROLIFERATIVE lesion)
- 35. **Atypical bathing suit**: ATYPICAL EPITHELIAL HYPERPLASIA (hyperplastic epithelial cells acquire atypia → disorganized cells with dark purple nuclei) (a PROLIFERATIVE lesion)
- 36. Atypical girl raising cancer crab: ATYPICAL EPITHELIAL HYPERPLASIA increases the risk of breast cancer
- 37. Excessive glandy sponges: SCLEROSING ADENOSIS (increased glandular tissue in lobules [due to proliferation of luminal spaces]) (a PROLIFERATIVE lesion)
- 38. **Sclerotic snake-ish hose**: in SCLEROSING ADENOSIS, the stroma contains increased fibrous tissue ("sclerosis")
- 39. **Crab handle**: SCLEROSING ADENOSIS increases risk of breast cancer)
- 40. Young woman ADDing FIBERS to bouquet: FIBROADENOMA (benign breast tumor) (occurs in young women [ages 15-35])
- 41. **Pregnant and wearing female jewelry**: FIBROADENOMAS are estrogen-responsive (size increases with high estrogen levels [pregnancy, lactation, pre-ovulation] and decreases with menopause)
- 42. **Rollerblade wheels**: FIBROADENOMAS are well-defined, rubbery, mobile masses
- 43. Happy face: FIBROADENOMAS are painLESS
- 44. **Stromal and gland-y concrete**: FIBROADENOMAS are a result of proliferation of BOTH (fibrous) stroma & (adenomatous) ducts → histology demonstrates benign-appearing stroma & epithelium-lined glandular spaces
- 45. **Compressing ducts**: on histology, stromal proliferation leads to compression of glandular spaces
- 46. Large pile of leaves: PHYLLODES tumors (BENIGN fibroepithelial tumors) (often very large)
- 47. **Older lady on roller skates**: PHYLLODES tumors typically present in older women (age 40s)
- 48. **Leafy pattern**: on histology, PHYLLODES tumors display leaf-like papillary projections

- 49. Roller skate wheels: PHYLLODES tumors are well-defined, painless, mobile masses (similar to fibroadenomas)
- 50. **Crabby rake**: PHYLLODES tumors are capable of malignant behavior
- 51. Papillary bush poking into slide lumen: INTRADUCTAL PAPILLOMAS (papillary tumors located in lactiferous ducts) (grow from duct wall into the lumen)
- 52. **Bloody water**: INTRADUCTAL PAPILLOMAS are the most common cause of bloody nipple discharge
- 53. **Twisted red hose**: in INTRADUCTAL PAPILLOMAS, bloody nipple discharge is a result of twisting of the vascular stalk (which connects papilloma to duct wall)
- 54. **Fibrous plant core** : on histology, INTRADUCTAL PAPILLOMAS display a fibrovascular core with papillary projections
- 55. Crab handle : INTRADUCTAL PAPILLOMAS can hide areas of cellular atypia or ductal carcinoma in situ \rightarrow risk of transformation into breast cancer
- 56. **Releasing milk**: GALACTORRHEA (bilateral milky nipple discharge unrelated to breastfeeding)
- 57. **Touching milk nipple**: GALACTORRHEA can be caused by mechanical nipple stimulation
- 58. Excessive prolactin baby bottles in pituitary purse: prolactinomas (prolactin-secreting tumors in anterior pituitary) can cause GALACTORRHEA (elevated prolactin levels → milky nipple discharge)
- 59. **Breaking double ropes**: D2 dopamine receptor antagonists (antipsychotics) can cause GALACTORRHEA (inhibition of inhibitory dopamine activity on anterior pituitary → elevated prolactin levels → milky nipple discharge)

Reproductive & GU 4.2 - Breast Cancer



- 1. Crab bra: breast cancer
- 2. "1" finger: breast cancer is the #1 most common cancer in women (and 2nd most common cause of cancer death)
- 3. "Family Farms": family history is a risk factor for breast cancer
- 4. **Oversized BRoccoli with 1 floret**: BRCA1 mutations are a significant risk factor for breast cancer
- 5. Oversized BRoccoli with 2 florets: BRCA2 mutations are a significant risk factor for breast cancer
- 6. Domino: BRCA1 and 2 are autosomal DOMINANT
- 7. Cancer fighting ribbons: BRCA1 and 2 are tumor suppressor genes
- 8. **1 ribbon already down**: BRCA mutations follow the "2-hit" hypothesis (patients inherit one mutated BRCA allele → breast cancer develops if the second allele is hit by a random somatic mutation during their lifetime)
- 9. **So FrauMany Varieties**: Li-Fraumeni syndrome is a risk factor for breast cancer (and many other tumors)
- 10. **Brain squash, bone-y zucchini, adrenal-ish fruits, breast-y gourds**: Li-Fraumeni syndrome is associated with tumors in bone, brain, and adrenal glands (in addition to breast cancer)
- 11. **Broken "Plea53" checkpoint gate**: Li-Fraumeni syndrome is caused by a mutation in the p53 tumor suppressor gene (functions as a checkpoint in the a cell cycle)
- 12. **Domino-patterned sign** : Li-Fraumeni syndrome is autosomal DOMINANT
- 13. Older lady with female jewelry & "paused" commercial: exposure to exogenous estrogen AFTER menopause is a risk factor for breast cancer
- 14. **Obese lady**: obesity is a risk factor for breast cancer (aromatase in adipose cells converts androstenedione to estrone → excess estrogen → increased cancer risk)
- 15. Early bloomers: early menarche is a risk factor for breast cancer
- 16. Late deciduous: late menopause is a risk factor for breast cancer
- 17. Non-fruit bearing: nulliparity is a risk factor for breast cancer
- 18. **Baby on "Don't Wait" sign** : older age at first pregnancy is a risk factor for breast cancer
- 19. **Holding baby at breast**: breastfeeding decreases the risk of breast cancer

- 20. **Oversized PROGESTERO-DOME**: some breast cancers overexpress PROGESTERONE receptors (PR)
- 21. **Holding HER 2 NEWborns**: some breast cancers overexpress the HER2/NEU proto-oncogene (an EGF receptor)
- 22. **Oversized female toothpick** : some breast cancers overexpress ESTROGEN receptors (ER)
- 23. Sad because of 3 empty plates: "Triple Negative" breast cancer (does not overexpress PR, ER, or HER2/NEU) has a poorer prognosis
- 24. **Ductal turnip with intact skin**: DUCTAL carcinoma in situ (DCIS) (does NOT invade basement membrane) (precursor to invasive ductal carcinoma) (cells arising from terminal ducts form a MASS that eventually fills the duct lumen)
- 25. Central skull: DCIS displays prominent central necrosis
- 26. **White speckles**: DCIS produces microcalcifications (visible on mammogram)
- 27. **Skull & crossbones shirt**: DCIS is most commonly diagnosed with mammography (specialized X-ray of the breasts)
- 28. Knife: DCIS & LCIS are primarily treated with surgery
- 29. **Lobular leaf**: lobular carcinoma in situ (LCIS) (precursor to invasive ductal & lobular carcinomas) (cells arising from terminal ducts migrate to fill lobular space) (on histology, cells with dark round nuclei form clusters within lobules) (NO palpable mass, NO calcifications on mammogram)
- 30. Knife: DCIS & LCIS are primarily treated with surgery

Reproductive & GU 4.2 - Breast Cancer



- 31. Crabbed-shirt kid popping out of ductal turnip: INVASIVE DUCTAL carcinoma (malignant cells have invaded through ductal basement membrane) (most common breast cancer)
- 32. **IMMOBILE summer squash**: INVASIVE DUCTAL carcinoma presents as a painless, FIXED (non-mobile), hard breast mass with irregular borders (most common in upper outer quadrant)
- 33. Squash with dimples & cratered top: INVASIVE DUCTAL carcinoma can present with skin dimpling or inverted nipples (tumor infiltrates suspensory [Cooper's] ligaments \rightarrow ligament fibrosis and shortening \rightarrow pulls skin/nipple inward)
- 34. **Skull & crossbones shirt**: INVASIVE DUCTAL carcinoma can be detected by mammography
- 35. **Spiky sun rays**: INVASIVE DUCTAL carcinoma displays a SPICULATED ("spiky") soft tissue mass on mammogram
- 36. **Tiny stars**: INVASIVE DUCTAL carcinoma displays clustered microcalcifications on mammogram
- 37. **Purple glands within pink stroma**: on histology, INVASIVE DUCTAL carcinoma displays nests of glandular tissue (hypercellular ducts with irregular dark purple cells) surrounded by dense fibrous stroma (fibrotic due to desmoplastic reaction)
- 38. Crabbed-shirt kid with oversized leaf masses: INVASIVE LOBULAR carcinoma (second most common breast cancer) (usually presents with palpable mass and densities on mammography
- 39. 2 leaf masses & scattered leaves : INVASIVE LOBULAR carcinoma is more likely to be BIIATERAL and present with multiple tumors
- $40. \ \mbox{\bf Single file line}:$ on histology, INVASIVE LOBULAR carcinoma displays orderly rows of cells
- 41. Enormous blood orange with thick dimpled peel:
- INFLAMMATORY breast cancer (presents with "peau d'orange" [erythematous, thickened, dimpled appearance of overlying skin due to lymphedema and inflammation])
- 42. **Thick skin**: on histology, INFLAMMATORY breast cancer displays dermal lymphatic invasion by poorly differentiated tumor cells
- 43. **kid throwing seeds**: INFLAMMATORY breast cancer metastasizes early (due to early invasion of lymphatic system)
- 44. "MID-JULY": MEDULLARY carcinoma
- 45. **Crowd of BLUE hats**: on histology, MEDULLARY carcinoma displays poorly-differentiated cells with significant lymphocytic infiltrate (small cells with prominent BLUE nuclei)

- 46. **Soft slobbery dog**: MUCINOUS carcinoma (presents with a soft, gelatinous well-circumscribed mass [due to spread of mucin into surrounding stroma])
- 47. **Slobber pool** : on histology, MUCINOUS carcinoma displays nests of tumor cells within large pools of extracellular mucus
- 48. **Tubular green onions**: TUBULAR carcinoma (histology displays well-formed tubules infiltrating stroma)
- 49. **Happy face**: TUBULAR carcinomas have an excellent prognosis (due to low grade)
- 50. **Passion fruit**: PAGET disease (malignant cells within the skin OVERLYING a breast carcinoma) (due to cells spreading through the lactiferous ducts and into the epidermis of the nipple)
- 51. **Eczematous leaf**: PAGET disease presents as an ulcerated eczematous skin lesion (begins a the nipple and spreads outward)
- 52. **Passion fruit seeds**: PAGET cells (malignant intraepithelial adenocarcinoma cells) (large cells with clear cytoplasm and dark nuclei)
- 53. **Passion fruit seeds invading epidermal dirt**: in PAGET disease, PAGET cells invade the epidermis of the nipple
- 54. **Passion fruit seeds sitting on lap**: PAGET disease can also present in the genital region (most commonly the vulva) ("EXTRAmammary paget's) (appears identical to disease of the breast)
- 55. **Happy face**: EXTRAmammary PAGET disease is NOT typically associated with an underlying malignancy
- 56. "Take a SAMPLE": Selective Estrogen Receptor Modulators (SERMS) (bind estrogen receptors → act as agonists OR antagonists depending on the specific tissue type)
- 57. "TIME to RELAX" near estrogen toothpick: breast cancers that overexpress ESTROGEN receptors can be treated with Tamoxifen or Raloxifene (SERMs with antagonist effects on estrogen receptors in the breast)
- 58. **Man with 2 floret broccoli**: male breast cancer usually occurs in men with BRCA2 mutations

Reproductive & GU 5.1 - Testicular Disorders & Cancer





- 1. **Orchid** : testes (before birth, normally descend from abdomen \rightarrow through inguinal canal \rightarrow into scrotum)
- 2. **Holding back tethered ball**: CRYPTORCHIDISM (incomplete descent of a testicle) (may be located anywhere along path of descent)
- 3. **ONE pocket pulled out** : in CRYPTORCHIDISM, the undescended testes is most commonly in the inguinal canal \rightarrow inguinal fullness or palpable mass (usually UNILATERAL)
- 4. **Unripened green fruit**: CRYPTORCHIDISM is common in premature infants
- 2 outturned pockets: CRYPTORCHIDISM associated with a genetic disorders is more likely to be BILATERAL (however, cryptorchidism is usually an ISOLATED finding)
- 6. **Calvin KLEIN FELT sweater**: KLINEFELTER syndrome can present with CRYPTORCHIDISM (also hypogonadism due to small firm hypofunctional testes)
- 7. "Y" collar with "X" argyle design : 47 XXY is the genotype in KLINEFELTER syndrome
- 8. **Trampled male trowels**: genetic disorders that cause abnormal sexual development or hypogonadism can present with CRYPTORCHIDISM (initial state of testicular descent is controlled by androgens and other hormones)
- 9. **Tetherball with cancer crab & mass**: CRYPTORCHIDISM increases the risk of testicular cancer (particularly in undescended testes, but also small risk in contralateral side)
- 10. **High temperature read on thermometer**: normal body temperature is higher than scrotal temperature
- 11. **Dried up infertility tree**: CRYPTORCHIDISM can present with infertility (exposure of undescended testis to higher temperature of the abdomen \rightarrow atrophy of Sertoli cells and seminiferous tubules)
- 12. **Twisted tetherball string**: CRYPTORCHIDISM can be complicated by testicular TORSION (since the undescended testicle in not properly secured inferiorly)
- 13. **Scalpel**: CRYPTORCHIDISM is treated with surgery to bring undescended testis into scrotum (orchiopexy) (decreases cancer risk)
- 14. **Worms in LEFT pocket** : increased pressure in left renal vein \rightarrow increased pressure in left testicular vein \rightarrow retrograde flow \rightarrow eventually dilation of pampiniform plexus in spermatic cord \rightarrow VARICOCELE on LEFT
- 15. Kidney reservoir attached to gutter system: LEFT KIDNEY is drained by left renal vein

- 16. **90° angle**: LEFT RENAL vein connects to LEFT TESTICULAR vein at a 90° angle (inefficient design for drainage) (angle is less acute on right side)
- 17. tree branch overlying gutter : SUPERIOR MESENTERIC ARTERY (coming off aorta) passes OVER LEFT renal vein \rightarrow potential for vein compression \rightarrow left sided varicocele
- 18. **Standing in pain**: VARICOCELE presents with aching pain in affected scrotum (most noticeable when standing and improved with lying supine)
- 19. Wormy infertility tree : chronic varicocele \to pressure in scrotum \to testicular atrophy/infertility
- 20. **Tether ball twisted around pole**: TESTICULAR TORSION (testes twist around the spermatic cord)
- 21. "VIRGINIA" : TESTICULAR TORSION is usually due to inadequate fixation of the testis to the tunica Vaginalis of the scrotal sac (congenital defect) \rightarrow free to rotate
- 22. **Twisted blue & red strings**: in TESTICULAR TORSION, rotation around spermatic cord \rightarrow compression of testicular vein \rightarrow testicular edema \rightarrow arterial compression
- 23. **Scalpel**: TESTICULAR TORSION is treated with emergency surgery (in order to prevent irreversible infarction of testis)
- 24. Clutching crotch & grimacing: TESTICULAR TORSION presents with acute scrotal pain
- 25. Green face : TESTICULAR TORSION presents with nausea & vomiting
- 26. **No CREAM cookies**: TESTICULAR TORSION presents with absent CREMasteric reflex (normally, pinching skin of upper thigh \rightarrow elevation of ipsilateral testis)
- 27. Water balloon & "VIRGINIA" pants: HYDROCELE (collection of serous fluid within the tunica VAGINALIS)
- 28. **Smiling**: HYDROCELE presents with painLESS swelling of the scrotum
- 29. **Persistently patent pants**: a COMMUNICATING HYDROCELE (congenital) results from incomplete obliteration of processus vaginalis (peritoneal tissue that travels with testes through inguinal canal during development) → fluid from peritoneal cavity can flow into tunica VAGINALIS
- 30. **Bacteria balloon**: acquired HYDROCELE (generally NONcommunicating) commonly occurs after an infection (epididymitis), torsion, trauma, or tumor
- 31. Glowing spot beneath balloon: HYDROCELES will transilluminate (a beam of light can pass through it) (in contrast to varicocele or tumor)

Reproductive & GU 5.1 - Testicular Disorders & Cancer





- 32. **Tetherball with cancer crab & mass**: TESTICULAR CANCER (most common malignancy in males between ages 15-35)
- 33. "GERMINATING" seeds: most testicular tumors are GERM cell tumors (divided into pure seminomas & NONseminomas)
- 34. **Sea-man superhero**: SEMINOMAS (most common testicular tumor) (identical to dysgerminomas of the ovary)
- 35. **Homogenously enlarged sperm whale**: SEMINOMAS present as homogenous testicular enlargement (as opposed to an isolated mass)]
- 36. **Purple sunny side up egg**: on histology, SEMINOMAS display a "fried egg" appearance (identical to ovarian dysgerminomas)
- 37. **β-hCG babysitter**: β-hCG is a tumor marker for SEMINOMAS (and ovarian dysgerminomas)
- 38. **Dehydrated LDH milk**: lactate dehydrogenase (LDH) is a tumor marker for SEMINOMAS (and ovarian dysgerminomas)
- 39. **Egg yolk**: YOLK SAC (endodermal sinus) tumors (NONseminomas) (similar to ovarian yolk sac tumor) (most common tumor in boys age <3)
- 40. **Egg chiller**: on histology, YOLK SAC tumors display Schiller-Duval bodies (papillary structures with central vessel)
- 41. "Alf's Fresh Produce" : α -fetoprotein (AFP) is a tumor marker for YOLK SAC tumors
- 42. **Terrariums**: TERATOMAS (NONseminomas) (similar to ovarian teratomas)
- 43. **Three-layered terrarium**: MATURE TERATOMAS contain cells from 3 germ layers (ectoderm, mesoderm, endoderm) (often includes hair & teeth)
- 44. Addition of cancer crab: MATURE TERATOMAS in the testicles can be benign or MALIGNANT (in contrast to ovary, where they are ALWAYS benin)
- 45. **Terrarium with amorphous rocks and escaping crabs**: IMMATURE TERATOMAS (contain poorly differentiated tissues) (malignant in both testicle and ovary)
- 46. **Frog EMBRYO**: EMBRYONAL CARCINOMAS (NONseminomas) (far more common in testes compared to ovaries)
- 47. **Bleeding skull shirt**: EMBRYONAL CARCINOMA appears as hemorrhagic masses with areas of necrosis
- 48. **Purple amorphous algae**: on histology, EMBRYONAL CARCINOMA displays sheets of atypical poorly differentiated cells
- 49. $\beta\text{-hCG}$ babysitter : $\beta\text{-hCG}$ is a tumor marker for EMBRYONAL CARCINOMAS

- 50. **CHOIR music box**: CHORIOCARCINOMAS (NONseminomas) (similar to placental trophoblastic tumor in females)
- 51. **Trophy case**: CHORIOCARCINOMAS in FEMALES are derived from chorionic villi (composed of 2 cell types: syncytiotrophoblasts and cytotrophoblasts
- 52. Falling CYTO-cycling & SYNCHRONIZED swimming trophies: TESTICULAR CHORIOCARCINOMAS contain disordered proliferations of both mononuclear CYTOtrophoblasts & multinucleated SYNCYTIOtrophoblasts
- 53. Hit in the head & bleeding : CHORIOCARCINOMAS disseminate hematogenously early in the disease (highly aggressive) \rightarrow frequently metastasize to lung or brain by the time of diagnosis
- 54. Large β -hCG babysitter : CHORIOCARCINOMAS are characterized by EXTREMELY high levels of β -hCG (similar to female choriocarcinomas)
- 55. **Tying large bowtie**: CHORIOCARCINOMAS can present with hyperthyroidism (due to extremely high β -hCG) (hCG and TSH have a common alpha subunit \rightarrow hCG binds TSH receptor on thyroid gland \rightarrow stimulate production of thyroid hormone)
- 56. Vases projected onto chest : CHORIOCARCINOMA can present with gynecomastia (extremely high β -hCG levels \rightarrow leydig cell dysfunction \rightarrow increased estrogen production)

Reproductive & GU 5.2 - Benign Prostatic Hyperplasia (BPH) & Prostate Cancer





- Old fire fighter squeezing light pole: benign prostatic hyperPLASIA (BPH) is an extremely common enlargement of the prostate seen in older men
- 2. "TRANSIT" sign: BPH is caused by hyperplasia of cells in the transitional (innermost) zone of the prostate (which surrounds urethra)
- 3. "Mid Day and Late Day" busses: the transitional zone of the prostate include the middle and lateral lobes, which surround the urethra (enlargement causes urinary obstruction)
- 4. **Alpha team volunteer opening dihydro hydrant** : 5-alpha reductase converts testosterone into the more potent dihydrotestosterone → prostatic epithelial and stromal hyperplasia → prostate enlargement
- 5. **Epithelial clovers with smooth pink leaves**: DHT stimulates an increase in the NUMBER of glandular epithelial and smooth muscle cells (hyperPLASIA) in the prostate (along with increased stroma)
- 6. Circular, smooth concrete slab: BPH forms smooth, symmetric enlargement of the prostate containing both stromal and epithelial components (asymmetry or nodularity should raise suspicion for malignancy)
- 7. **Struggling to open valve**: BPH commonly causes hesitancy (difficulty initiating micturition)
- 8. **Weak pressure hose**: BPH commonly causes weak urinary stream, or "dribbling"
- 9. **Urgent situation**: BPH commonly causes urgency (an intense feeling the need to void)
- 10. **Multiple little puddles**: BPH commonly causes increased frequency of micturition due to incomplete voiding
- 11. **Sleepy fireman**: BPH commonly causes nocturia (waking up to urinate) due to incomplete voiding during the day
- 12. Full bladder tank: ureteral obstruction due to BPH can cause urinary retention and bladder distention
- 13. **Dilated yellow hose from kidney tank**: chronic urinary obstruction from BPH can cause bilateral ureteral dilation and hydronephrosis
- 14. Cracked kidney water tank: chronic urinary obstruction from BPH can cause post-renal azotemia, AKI, and even renal failure
- 15. **UTI cup in truck**: chronic urinary retention in BPH can lead to recurrent urinary tract infections (though rare)
- 16. **Smooth ballooning hose**: on digital rectal exam, BPH will feature smooth, symmetric enlargement of the prostate without nodules or tenderness
- 17. **Blowing out alpha candle**: alpha-1 antagonists (such as terazosin) improve BPH by causing relaxation of smooth muscle surrounding the prostatic urethra (no effect on prostate VOLUME)

- 18. **Asteride asteroid debris**: 5-alpha reductase inhibitors (finasteride, dutasteride) inhibit the conversion of testosterone to DHT in prostatic tissue→ decrease glandular and stromal volume→ decrease prostate size
- 19. **#1 fire fighter fan**: prostate adenocarcinoma is the most common malignancy and the SECOND most common cause of cancer death in males
- 20. **Family affair**: family history of prostate carcinoma (especially in first degree relatives) is a risk factor for prostate carcinoma development
- 21. **Black firefighter with cancer crab** : prostate is more common in African American males
- 22. **Seasoned smoking fire fighter**: smoking is a risk factor for prostate adenocarcinoma
- 23. **Staying out in peripheral zone**: prostate cancer is often asymptomatic due to its development in the PERIPHERAL zone of the prostate
- 24. **POSTal box** : prostate cancer most commonly develops in the POSTERIOR lobe of the peripheral zone→ rarely causes urinary symptoms
- 25. **Boosted up one side of knotty tree**: on DRE, prostate CANCER features asymmetrical enlargement, nodule formation, and areas of induration (as opposed to firm, symmetrical enlargement in BPH)
- 26. **Climbing bony ladder**: prostate cancer metastasizes early, especially to the vertebral column, via lymphatic and hematogenous spread
- 27. **Painful fall onto back**: prostate cancer metastasis to the vertebral column typically presents with back pain
- 28. **Osteobuilder holding bony ladder**: prostate cancer forms osteoBLASTIC (bone forming) metastases
- 29. "PSA" awareness sign: prostate specific antigen (PSA) is produced by prostatic epithelial cells and is used to monitor for recurrence in men with a history of prior prostate cancer (asymptomatic PSA screening is controversial)
- 30. **Irregular puddle**: histologically, prostate cancer has well defined gland composed of cells with dark cytoplasm and large nuclei
- 31. "GaRdener's Hardware continuous service": continuous administration of GnRH agonists (leuprolide) inhibits secretion of LH and FSH→ decreased androgens (pharmacologic treatment of prostate cancer)
- 32. Cracked window over male symbol tools : continuous GnRH agonist administration decreases androgens→ inhibition of prostate cancer growth (prostate cancer is androgen dependent)

Reproductive & GU 5.3 - Bladder Cancer & Penile Disorders



- 1. **Transitional window treatment**: (urothelium) urothelium, the epithelial lining from the renal pelvis to the urethra, is stratified and changes shape as it contracts or expands (as with bladder emptying)
- 2. **Bladder rock with ureter sprouts**: urothelial carcinoma can form anywhere along the urothelial tract (renal pelvis to superior urethra)
- 3. **Multifocal hermit crabs**: urothelial carcinoma tends to be multifocal (most likely due to diffusion of carcinogens or metastases throughout the urine)
- 4. **Spotty transitional curtains**: histologically, urothelial cancer resembles normal urothelium but with hypercellularity, hyperchromasia, and loss of cellular polarity
- 5. Gloves, leather belt, and hard hat to mix blue paint: exposure to aromatic amines or beta-naphthylamine (as in the rubber, plastic, dye, paint, and leather industries) is a risk factor for urothelial carcinoma (metabolites of these products are excreted in the urine)
- 6. Smoker: smoking is a risk factor for urothelial cancer
- 7. **Cyclops by hermit crabs**: treatment with the antineoplastic and immunosuppressant cyclophosphamide (alkylating agent) is a risk factor for urothelial carcinoma
- 8. New squamous tile floors: squamous cell epithelium
- 9. **Burned squamous tile floor** : chronic inflammation of transitional epithelium→ metaplastic change to tougher squamous epithelium
- 10. **Swordfish clock by fire**: Schistosoma haematobium infection causes chronic infection and inflammation of the bladder urothelium and its venous plexus→ squamous metaplasia
- 11. **Smoking squamous sailor**: smoking is a risk factor for squamous cell carcinoma of the bladder and urinary tract
- 12. **UTI cup**: inflammation from chronic urinary tract infections is a risk factor for squamous cell carcinoma of the bladder and urinary tract
- 13. **Crushed berries dripping red**: both urothelial and squamous cell carcinoma of the bladder and urinary tract commonly present with PAINLESS hematuria (gross or microscopic)
- 14. "**Drains to Atlantic**": the allantois is a fetal structure that connects the yolk sac to the urogenital sinus and is normally obliterated to form the urachus
- 15. **Raking clogged leaves to open allantoic drain**: failure of the allantois to completely involute results in a PATENT urachus→ abnormal open conduit from bladder to umbilicus
- 16. **Wet leaf on belly**: the open connection from the bladder to the umbilicus with a patent urachus presents with leakage of urine from the umbilicus in newborns
- 17. **Crab on squamous door with phallic canopy**: squamous cell carcinoma is the most common penile cancer and is more common in developing countries
- 18. **Pill bugs by phallic door**: human papillomavirus infection (especially high risk strains 16 and 18) is a risk factor for penile squamous cell carcinoma

- 19. Weiner wizard hat: HIV infection is a risk factor for penile squamous cell carcinoma
- 20. **Dirty flesh colored canopy**: uncircumcised males with a history of poor hygiene or phimosis (fibrosed foreskin that can't be retracted from the glans) have a higher risk of penile carcinoma
- 21. Captain Johnson smoking : smoking is a risk factor for penile squamous cell carcinoma
- 22. **Old salty seamen under canopy**: penile squamous cell carcinoma typically presents in men over 60 with a painless lesion on the glans or foreskin
- 23. "Canine InSide": carcinoma in situ (CIS) involves full thickness epithelial expansion of malignant cells WITHOUT invasion of the basement membrane (NO malignant cells in dermis)
- 24. **Crusty red stone under bow**: Bowen disease is a penile carcinoma in situ that typically presents with a crusting, oozing erythematous plaque on the shaft of the penis
- 25. **Beefy red doormat**: Erythroplasia of Queyrat is a penile carcinoma in situ that typically presents as a velvety red lesion on the glans penis (10-30% progress to squamous carcinoma)
- 26. **Bowenoid bows on package**: Bowenoid papulosis presents with multiple red papules throughout the penis and RARELY progresses to carcinoma
- 27. **Buried spade tool**: hypospadias is a urethral opening on the VENTRAL surface of the penis and is the most common congenital penile disorder
- 28. **Unlatched general tool basket**: hypospadias develops due to incomplete fusion of the urogenital (urethral) folds during development
- 29. **Dilated pipe**: hypospadias can cause urinary outlet obstruction (can lead to post-renal AKI and hydronephrosis in severe cases)
- 30. **Unplanted berry orchid**: epispadias is associated with an increased incidence of cryptorchidism (undescended testicle)
- 31. **Erecting spade tool**: epispadias is an abnormal urethral opening on the DORSAL surface of the penis
- 32. **Jimmy's tuber garden**: epispadias results due to abnormal positioning of the genital tubercle during development
- 33. **Red fanny sack**: epispadias is associated with bladder exstrophy, an abdominal wall defect that results in opening of the interior bladder mucosa to the environment
- 34. **Droopy Peyronie pine**: Peyronie disease is bending and contracture of the penile shaft caused by fibrosis
- 35. **Alabama slamma**: Peyronie disease is caused by abnormal collagen, fibroblasts, and elastic fibers of the tunica albuginea (fibrous tissue surrounding penis)
- 36. Painful crotch shot: contractures in Peyronie disease cause penile pain
- 37. Hose no goes: Peyronie disease can cause erectile dysfunction

Blood & Coagulation 1.1 - Microcytic Anemia: Overview & Thalassemias



- 1. Micro Polo: MICROcytic anemia
- 2. "T.A.I.L.S.": microcytic anemia is caused by T.A.I.L.S (Thalassemia, Anemia of chronic disease, Iron deficiency, Lead, Sideroblastic Anemia)
- 3. "< 80" teapot : MICROcytic anemia is defined as mean corpuscular volume (MCV) less than 80)
- 4. **Purple tea bag**: reticulocytes (immature RBCs with "net like" chromatin)] remain normal in microcytic anemia (due to inability to increase hemoglobin production)
- 5. "<3": in microcytic anemia, the reticulocyte count is low (less than 3%)
- 6. **Small pale pink teacups** : MICROcytic anemia (RBCs with microcytosis and hypochromia)
- 7. "Bull's eye" saucers : microcytic anemia causes target cells (excess membrane on microcytic RBCs \rightarrow folded cell membranes \rightarrow target appearance)
- 8. **Broken CHALICEmia**: thalassemia (microcytic anemia due to dysfunctional hemoglobin production, can be due to defective alpha or beta globins)
- 9. " α " and " β " handles : normal hemoglobin (A1) is composed of 2 alpha and 2 beta subunits
- 10. "16" flag: alpha globin chains are encoded on chromosome 16
- 11. Two α beads on each beard strand : each chromosome has 2 alpha globin alleles (for a total of 4 alleles)
- 12. **Asian territory**: ALPHA thalassemias are most common in Southeast Asia and Sub-Saharan Africa
- 13. "11" flag: beta globin chains are encoded on chromosome 11
- 14. **Mediterranean merchant**: BETA thalassemias are most common in Greeks, Italians, and Africans
- 15. Luck symbol: ALPHA thalassemia MINIMA is asymptomatic
- 16. **Two red beads with pink-clothes**: ALPHA thalassemia TRAIT (caused by 2 alpha subunit deletions)
- 17. Pink robe: ALPHA thalassemia TRAIT causes MILD anemia

- 18. "TRANS-AFRICA" map: ALPHA thalassemia TRAIT in the TRANS configuration is most common in Africans (1 alpha subunit deletion on each of 2 DNA strands)
- 19. **Pink Asian Cis-ters**: ALPHA thalassemia TRAIT in the CIS configuration is most common in Southeast Asians (2 alpha subunit deletions on the same DNA strand)
- 20. Large lychee bowel: ALPHA thalassemia TRAIT (and BETA thalassemia MINOR) may cause an INCREASED RBC count
- 21. **Pale pink lychee**: ALPHA thalassemia TRAIT causes microcytic anemia with hypochromic RBCs)(usually mild anemia, usually Asymptomatic)
- 22. **3 red beads**: Hemoglobin H disease (alpha thalassemia INTERMEDIA)(caused by 3 alpha subunit deletions)
- 23. **Beta "H" war hammer** : Hemoglobin H is composed of a tetramer of BETA globin chains
- 24. Blue face : hemoglobin H has a strong affinity for oxygen \to decreased oxygen delivery to tissues \to hypoxia
- 25. **Fast cavalry**: hemoglobin H migrates faster than normal hemoglobin (A1) on electrophoresis
- 26. Lychee seeds : hemoglobin H precipitates \rightarrow inclusion bodies that damage RBC membranes \rightarrow EXTRAVASCULAR hemolysis
- 27. **Falling lychee** : Hemoglobin H disease causes microcytic anemia with hypochromic RBCs (moderate to severe anemia)
- 28. **No alpha beads in hair**: ALPHA thalassemia MAJOR (due to deletions in all 4 alpha globin genes)
- 29. **4 gamma clasps** : ALPHA thalassemia MAJOR causes the formation of hemoglobin Bart's (tetramer of GAMMA subunits)
- 30. Blue face : hemoglobin Bart's has a high oxygen affinity \to minimal oxygen delivery to tissues \to hypoxia
- 31. **Red swollen doll**: hemoglobin Bart's causes hydrops fetalis (precipitation of hemoglobin Bart's → hemolysis → severe hypoxia → high-output heart failure → total body edema [hydrops] in fetus)
- 32. Lychee seeds : hemoglobin H precipitates \to inclusion bodies that damage RBC membranes \to EXTRAVASCULAR hemolysis

Blood & Coagulation 1.1 - Microcytic Anemia: Overview & Thalassemias



- 33. **SPICES** : mutation to the BETA globin gene can cause a mRNA splicing defect \rightarrow fewer subunits produced
- 34. Corked CODON bottles : mutation to the BETA globin gene can cause a premature stop codon \rightarrow no subunits produced
- 35. One red bead: BETA thalassemia MINOR (due to a mutation in ONE beta allele)
- 36. Pink robe: BETA thalassemia MINOR causes mild anemia
- 37. Cherry pits : in BETA thalassemia, excess ALPHA subunits precipitate ightarrow inclusion bodies
- 38. Discarded " α " cherry stems : excess ALPHA subunits (precipitate in BETA thalassemia)
- 39. **Cherries in macro-CAGE**: BETA thalassemia causes EXTRAvascular hemolysis (precipitation of ALPHA subunits → inclusion bodies that damage RBC membranes → hemolysis in the spleen)
- 40. **Sleeping baby & "F" band**: BETA thalassemia MINOR causes production of HbF (alpha & gamma globins)
- 41. **Two adults & "A2" band**: BETA thalassemia MINOR causes increased production of HbA2 (alpha & delta globins)
- 42. **2 red beads with 2 spices**: BETA thalassemia INTERMEDIA (caused by mild SPLICING defects in BOTH beta genes)(moderate anemia)

- 43. Losing BOTH beta beads: BETA thalassemia MAJOR (caused by COMPLETE deficiency of BOTH beta genes)(severe anemia)
- 44. Liver & spleen cowhide: BETA thalassemia MAJOR causes extramedullary hematopoiesis in liver and spleen (chronic hypoxemia \rightarrow kidneys release erythropoietin \rightarrow hematopoiesis in liver and spleen \rightarrow hepatosplenomegaly)
- 45. "Chipmunk" helmet: BETA thalassemia MAJOR causes extramedullary hematopoiesis in bone (hematopoiesis expands from medulla into bone cortex → expansion of facial bones ["chipmunk facies"])
- 46. "Crew cut" helmet : BETA thalassemia MAJOR causes extramedullary hematopoiesis in skull → expansion of skull bones → "crew cut" appearance)
- 47. **Red tear drops**: BETA thalassemia MAJOR is associated with dramatic poikilocytosis (abnormal RBC morphology including microcytes, target cells, and teardrop cells)
- 48. **Two adults & "A2" band** : BETA thalassemia MAJOR causes increased production of HbA2 (alpha & delta globins)
- 49. **Upright baby & large "F" band**: BETA thalassemia MAJOR causes production of large amounts of HgF (alpha & gamma globins)
- 50. Falling "A" compass: in BETA thalassemia MAJOR, a HbA1 band is absent
- 51. "Fe" weight: BETA thalassemia MAJOR leads to iron overload (secondary hemochromatosis)(primarily due to multiple transfusions)
- 52. **Plastic bone**: BETA thalassemia MAJOR can be complicated by aplastic crisis (infection with Parvovirus B19 \rightarrow decreased production of RBCs by bone marrow \rightarrow rapid decrease in hemoglobin and hematocrit [very problematic in severe thalassemia due to short lifespan of RBCs])
- 53. Flag with red string: BETA thalassemia MAJOR is treated with chronic RBC transfusions
- 54. **Mosquito net**: hemoglobinopathies (thalassemias, sickle disease, etc) confer a degree of protection against malaria (abnormal RBCs are more prone to lysis)

Blood & Coagulation 1.2 - Iron Deficiency Anemia & Anemia of Chronic Disease





- 1. Dwarf den: duodenum (location of iron absorption)
- 2. **iron IN buckets in the villous plants**: iron is stored in duodenal enterocytes in the form of FERRITIN
- 3. "Iron Porter": iron is transported from the enterocyte into the blood via the FERROPORTIN channel
- 4. Red dwarf with iron TRANSporter : in the bloodstream, iron is bound to TRANSFERRIN
- 5. **Iron IN bucket in cage**: iron is stored INside of macrophages in the form of ferritin (in bone marrow)
- 6. **Red dwarf measuring iron IN bucket**: small amounts of ferritin are present in the serum (reflects the total amount of stored ferritin, can be used as a surrogate marker for iron reserves)
- 7. **Key dwarf with overloaded liver satchel**: the liver senses increased iron stores and releases hepcidin (LOCKS DOWN iron absorption and release of iron from macrophages)
- 8. Key locking iron-porter : hepcidin degrades ferroportin \rightarrow blocks release of iron from enterocytes \rightarrow decreased iron absorption
- 9. **Iron locked INSIDE cage**: HEPCIDIN inhibits release of iron from macrophages
- 10. Setting down iron tool next to Snow White: iron deficiency anemia
- 11. Celery bouquet : a vegetarian diet (low in iron can cause iron deficiency anemia
- 12. **Red cloth**: heavy menstrual bleeding, pregnancy, delivery, and lactation can cause iron deficiency anemia in reproductive age women
- 13. Colonic cancer crabs : GI cancer can cause iron deficiency anemia (via chronic occult bleeding)
- 14. **Polypy mushrooms**: benign hamartomatous polyps can cause iron deficiency anemia (via chronic occult blood loss)
- 15. **Stomach burial site**: erosive gastritis and gastric ulcers can cause iron deficiency anemia (via chronic occult bleeding)
- 16. **Hook pulley**: hookworms (Ancylostoma, Necator) and whipworms (T. trichiura) can cause iron deficiency anemia (via occur chronic blood loss)
- 17. **Stomach pot on fire** : chronic atrophic gastritis (H pylori-related and autoimmune) can cause iron deficiency anemia (inflammation and atrophy of gastric mucosa → decreased acid production → decreased release of iron from food)
- 18. Helicopter hat: H pylori associated gastritis
- 19. Antibody sticks: autoimmune gastritis
- 20. **SEAL**: CELiac disease can cause iron deficiency anemia (small bowel inflammation and injury \rightarrow decreased absorption of iron)
- 21. **Iron IN bucket on decreasing scale**: symptoms and signs of iron deficiency occur only after ferritin in the bone marrow is depleted
- 22. Broken heme wheels : decreased availability of iron leads to anemia (deceased iron \rightarrow decreased heme formation \rightarrow decreased hemoglobin)
- 23. Normal flowers: iron deficiency anemia is initially NORMOcytic
- 24. **Small shriveled flowers**: iron deficiency anemia later becomes MICROCYTIC

- 25. Lack of iron IN bucket: in iron deficiency anemia, FERRITIN stores are low (in both bone marrow and serum)
- 26. Elevated empty iron TRANSporter: in iron deficiency anemia, TRANSFERRIN is ELEVATED (due to increased transferrin production)
- 27. Empty iron TRANSporter : in iron deficiency anemia, transferrin saturation is LOW (due to decreased iron) → INCREASED total iron binding capacity [TIBC])
- 28. Pale & sleeping: iron deficiency anemia can present with fatigue, weakness, and skin pallor
- 29. White gloves and eye sash: in anemia, skin pallor is most notable in the palmar creases and eye conjunctiva $\,$
- 30. **Trembling dwarf heart**: severe anemia can cause a hyperdynamic state (low oxygen carrying capacity → increased cardiac output)(manifests as tachycardia and heart palpitations)
- 31. Falling plumbing tools: iron deficiency can cause Plummer-Vinson syndrome (dysphagia, esophageal webs, iron deficiency)
- 32. **Smooth red lips**: iron deficiency can cause atrophic glossitis (smooth red tongue)
- 33. **Inflamed mouth corners**: iron deficiency can cause angular cheilitis (inflamed corners of mouth)
- 34. **COILS popping out of curling thin mattress** : iron deficiency can cause KOILonychia (thin, brittle, "spoon-shaped" nails)
- 35. Magpie eating "ice": iron deficiency can cause pagophagia (inappropriate drive to eat ice)
- 36. Pale old hag with grandfather clock: anemia of chronic disease
- 37. **Bubbling cauldron**: anemia of chronic disease is caused by inflammation (leads to inhibition of iron release)
- 38. CytoCOINS: in anemia of chronic disease, underlying inflammation leads to an increase in CYTOKINES (e.g. IL-6)
- 39. **Key dwarf**: in anemia of chronic disease, HEPCIDIN is released from hepatocytes (locks iron INside of cells) due to an increase in cytokines
- 40. **Iron buckets IN macroCAGES**: in anemia of chronic disease, FERRITIN is increased (stored in macrophages in bone marrow)
- 41. Scattered iron tools: in anemia of chronic disease, serum IRON is low
- 42. Tumbling iron TRANSporter: in anemia of chronic disease,
- TRANSFERRIN is low (and therefore total iron binding capacity [TIBC] is low)
- 43. **Normal pink apples**: anemia of chronic disease is initially NORMOcytic
- 44. **Small shriveled pink apples** : anemia of chronic disease later becomes MICROcytic
- 45. **Fiery cauldron**: chronic inflammatory conditions are the most common cause of anemia of chronic disease (i.e. rheumatoid arthritis, inflammatory howel disease)
- 46. Inflammatory bone lanterns: rheumatoid arthritis
- 47. Fiery intestines: inflammatory bowel disease
- 48. Cancer crab: malignancy can cause anemia of chronic disease
- 49. XXX elixirs: alcohol can cause anemia of chronic disease



Blood & Coagulation 2.1 - Extravascular Hemolysis: Overview & RBC Membrane Defects



- 1. Spleen tornado: EXTRAvascular hemolysis
- 2. **Tomatoes sucked-up by tornado**: in EXTRAVASCULAR hemolysis, RBCs are destroyed in spleen (causes NORMOcytic anemia)
- 3. Stuck in fence slats: misshapen RBCs become trapped in the Cords of Billroth
- 4. **Caught in macroCAGEs**: RBCs trapped in the Cords of Billroth are phagocytosed by splenic macrophages (part of the reticuloendothelial system)
- 5. **Hemoglobin hubcaps stuck in macroCAGE**: in EXTRAvascular hemolysis, RBC digestion occurs inside the splenic macrophage → hemoglobin remains intracellular (no hemoglobinemia or hemoglobinuria)
- 6. **Uncollared billy goat**: in EXTRAvascular hemolysis, intracellular hemoglobin is converted to UNCONJUGATED bilirubin
- 7. **Yellow rain coat** : excessive bilirubin causes jaundice (occurs in EXTRAVASCULAR hemolysis)
- 8. **Peeing yellow goat**: excessive bilirubin leads to increased URObilinogen in the urine and stool (high bilirubin \rightarrow more bilirubin converted to URObilinogen in the GI tract \rightarrow more URObilinogen reabsorbed from GI tract \rightarrow increased urinary URObilinogen)
- 9. **SeaGALL**: excessive bilirubin can lead to pigmented gallstones (occurs in EXTRAvascular hemolysis)
- 10. Flying dehydrated milk: EXTRAvascular hemolysis can can cause elevated serum lactate dehydrogenase (LDH)(due to LDH released from digested RBCs)
- 11. **Flying purple tea bags**: in hemolytic anemia, the reticulocyte count is elevated (>3%) (due to increased RBC production by the bone marrow)
- 12. **Expanding red tornado** : chronic EXTRAvascular hemolysis leads to expansion of the red pulp in the spleen \rightarrow splenomegaly
- 13. "The Normans": NORMOcytic anemia (mean corpuscular volume [MCV] between 80-100)
- 14. **Spherical water tank**: hereditary spherocytosis (causes EXTRAvascular NORMOcytic hemolytic anemia)(due to defects in proteins that anchor the cell membrane to the cytoskeleton)
- 15. Rainbow SPECTRUM bands: SPECTRIN adheres RBC cell membrane to internal cytoskeleton
- 16. **Tarp**: external phospholipid bilayer of the RBC cell membrane (attached to the cytoskeleton via spectrin)
- 17. **Ripped SPECTRAL band**: mutations in SPECTRIN can cause hereditary spherocytosis (leads to weak, inflexible RBCs)
- 18. **Anchor letting go**: mutations in ANKYRIN (membrane structural protein associated with spectrin) can cause hereditary spherocytosis
- 19. Dominoes: hereditary spherocytosis is autosomal DOMINANT
- 20. Small sphere compared to normal water tank: in hereditary spherocytosis, RBCs lose vesicles of membrane in the splenic cords of Billroth (splenic conditioning) → small RBCs and increased RBC distribution width (RDW)

- 21. **Brick bouncing off metallic tank**: in hereditary spherocytosis, overstretched cell membrane leads to decreased membrane flexibility → phagocytosis by splenic macrophages → EXTRAvascular hemolysis
- 22. **Water bursting**: hereditary spherocytosis can be diagnosed with the osmotic fragility test (acidified glycerol lysis test)(overstretched RBCs lyse when placed in dilute solutions [due to inability to tolerate swelling of cell from osmosis])
- 23. Concentrated hemoglobin hubcaps: hereditary spherocytosis causes an increased Mean Corpuscular Hemoglobin Concentration (MCHC)(normal hemoglobin amount but decreased cell volume → increased concentration)
- 24. **Slobbery swollen chew toy**: hereditary spherocytosis can cause hydrops fetalis (severe anemia → high-output heart failure → total body edema [hydrops] in fetus)
- 25. **Plastic bone**: hereditary spherocytosis can be complicated by aplastic crisis (infection with Parvovirus B19 → decreased production of RBCs by bone marrow → rapid decrease in hemoglobin and hematocrit [very problematic in HS due to short lifespan of RBCs])
- 26. Elliptical red trailer: hereditary ELLIPTOCYTOSIS (due to mutations that case RBC cytoskeleton to lose its elastic recoil)
- 27. **Ripped SPECTRAL band**: mutations in SPECTRIN can cause hereditary ELLIPTOCYTOSIS
- 28. Dominoes: hereditary ELLIPTOCYTOSIS is autosomal dominant
- 29. **Window nets**: hereditary ELLIPTOCYTOSIS increases protection against malaria
- 30. "Private Property": pyruvate kinase deficiency (enzyme deficiency that causes EXTRAvascular hemolysis and NORMOcytic anemia)
- 31. **Shy kid**: pyruvate kinase deficiency is autosomal recessive (pyruvate kinase converts phosphoenolpyruvate into pyruvate, which is required for glycolysis)
- 32. **Losing three "P" batteries**: pyruvate kinase deficiency leads to insufficient ATP production \rightarrow loss of concentration gradient in RBC membrane \rightarrow loss of potassium and water \rightarrow defective membrane architecture
- 33. **Stuck in fence slats**: in pyruvate kinase deficiency, RBCs with decreased membrane flexibility become trapped in splenic Cords of Billroth → phagocytosis by macrophages → EXTRAvascular hemolysis
- 34. **Bumpy bonnet**: in pyruvate kinase deficiency, RBCs appear as burr cells (bumpy cell membrane due to the stiff cell architecture)(also called echinocytes)
- 35. **baby doll**: pyruvate kinase deficiency presents with hemolytic anemia in the newborn period, then splenomegaly later in life
- 36. Sharp farming implements: splenectomy is a treatment for EXTRAvascular hemolysis

Blood & Coagulation 2.2 - Intravascular Hemolysis: Overiew & Paroxysmal Nocturnal Hemoglobinuria (PNH)



- 1. **Torn tires on vascular road** : in INTRAVASCULAR hemolysis, RBCs lyse INSIDE blood vessels
- Leaking dehydrated milk : in INTRAvascular hemolysis, lysed RBCs release lactate dehydrogenase (LDH) directly into bloodstream → elevated serum LDH
- 3. **Hubcaps on vascular road**: INTRAvascular hemolysis leads to increased serum hemoglobin
- 4. Broken "HAPpy stay" sign : in INTRAvascular hemolysis, haptoglobin binds free hemoglobin → haptoglobin/hemoglobin complex is cleared from circulation by liver macrophages → decreased serum haptoglobin
- Hubcaps in draining water: INTRAvascular hemolysis leads to hemoglobinURIA (as a result of hemoglobinEMIA) (results in degradation of urine hemoglobin into heme or methemoglobin → brown urine)
- 6. **Rusty necrotic drainpipe**: severe INTRAvascular hemolysis can cause acute tubular necrosis (ATN) (heme released from lysed RBCs → tubular obstruction, direct toxicity, and vasoconstriction → kidney injury)
- 7. **Broken kidney window**: intravascular hemolysis can cause acute kidney injury (AKI) due to ATN
- 8. **Uncollared billy goat**: hemolysis (INTRA- and EXTRAvascular) leads to UNconjugated hyperbilirubinemia (due to breakdown of heme)
- Peeing goat : hyperbilirubinemia leads to increased URObilinogen in the urine and stool (high bilirubin → more bilirubin converted to URObilinogen in the GI tract → more URObilinogen reabsorbed from GI tract → increased urinary URObilinogen)
- 10. **SeaGALL**: hyperbilirubinemia can lead to pigmented gallstones (occurs in INTRA- and EXTRAvascular hemolysis)
- 11. **Holding up reticulated tea bag**: in hemolysis (INTRA- and EXTRAvascular), the reticulocyte count is elevated (>3%) (due to increased RBC production by the bone marrow)
- 12. "NORMAN": hemolysis (INTRA- and EXTRAvascular) usually causes a NORMOcytic anemia (MCV 80-100)

- 13. **Peaceful Night Hotel**: Paroxysmal Nocturnal Hemoglobinuria (PNH)(an ACQUIRED cause of INTRAvascular hemolysis)
- 14. "PIG"-A anchored to roof: the PIG-A gene codes for the first step in production of GPI anchor protein (holds other proteins to cell surface)
- 15. **Broken "X" anchor**: PNH is caused by an ACQUIRED mutation in the PIGA gene (located on the X chromosome)
- 16. Falling route "55" & "59": in PNH, the deficient GPI anchor protein leads to decreased CD 55 and CD 59 (complement inhibitor proteins) anchored to cell surface
- 17. **COMPLIMENTary breakfast**: in PNH, complement activation leads to INTRAvascular hemolysis (decreased CD 55 & CD 59 on RBC surface → activation of complement pathway including membrane attack complex (MAC) → RBC lysis
- 18. **Nearly empty pan**: PNH can cause pancytopenia (due to deficiency of GPI anchor protein on ALL hematopoietic cell lines)
- 19. **Dark liquid streaming from motel**: PNH causes nocturnal hemoglobinuria (dark urine in AM)
- 20. **Breathing into paper bag**: in PNH, dark urine in the AM is a result of mild respiratory acidosis during sleep (a normal process, but in PNH is sufficient to initiate complement activation)
- 21. Clusters of fibrotic sticks: PNH is associated with an increased risk of large vein thrombosis (especially abdominal and cranial veins
- 22. Water meters: PNH is diagnosed using flow cytometry (identifies deficiency of CD 55 & CD 59 proteins on cell surface)



Blood & Coagulation 2.3 - G6PD Deficiency & Autoimmune Hemolytic Anemia (AIHA)



- 1. "Norman's Motel": NORMOcytic anemias (includes G6PD deficiency and autoimmune hemolytic anemia)
- 2. **"66 ProDuce"** : glucose-6-phosphate dehydrogenase (G6PD)(rate limiting step in the pentose phosphate pathway)
- 3. **3 "P" stops**: Pentose Phosphate Pathway (responsible for synthesis of fatty acids and cholesterols; generates NADPH)
- 4. Scratched Route 66 sign: G6PD deficiency
- Dropping NADPH electrolyte drink: G6PD deficiency results in decreased production of NADPH (due to blocked pentose phosphate pathway)
- Decreasing "glute" exposure: G6PD deficiency results in decreased active glutathione (functions to prevent RBC from oxidative damage, requires reduction by NADPH in order to be recycled)
- 7. **Sparks from rusty hinges**: in G6PD deficiency, decreased glutathione results in increased exposure to oxidative damage → INTRA- and EXTRAvascular hemolysis at times of physiologic stress (e.g. infection)
- 8. Receding behind "X" gate: G6PD deficiency is X-linked recessive
- 9. **Oxidized hubcaps**: in G6PD deficiency, hemoglobin is exposed to oxidative stress (due to decreased glutathione) → clumps of denatured, insoluble hemoglobin → form inclusion bodies (Heinz bodies)
- 10. **Watermelon seeds**: in G6PD deficiency, Heinz bodies are visualized as small dots in the cytoplasm → damage to RBC membrane
- 11. Watermelon bites: G6PD deficiency causes 'bite cells' on blood smear (a result of splenic macrophages removing Heinz bodies and damaged membranes from the RBC)
- 12. **Tomatoes in macroCAGE** : in G6PD deficiency, damaged RBCs eventually undergo phagocytosis by macrophages in the spleen → EXTRAvascular hemolysis
- 13. **Broken tomatoes on ground**: in G6PD deficiency, some damaged RBCs undergo INTRAvascular hemolysis (less prominent than extravascular hemolysis)
- 14. **Brown exhaust liquid**: in G6PD, hemolysis presents with dark urine (due to elevated bilirubin)
- 15. **Yellow jacket**: in G6PD, hemolysis presents with jaundice (due to elevated bilirubin)
- 16. **Clutching side**: in G6PD, hemolysis presents with abdominal or back pain (due to renal injury)
- 17. **Bacterial veggies**: infection can precipitate hemolysis in G6PD deficiency
- 18. Deputy: dapsone can precipitate hemolysis in G6PD deficiency
- 19. **Stinky sulfa eggs**: trimethoprim-sulfamethoxazole can precipitate hemolysis in G6PD deficiency
- Sack of beans: fava beans can precipitate hemolysis in G6PD deficiency

- 21. Mosquito net: G6PD deficiency confers protection from malaria
- 22. "COLD" drinks: COLD autoimmune hemolytic anemia (AIHA)
- 23. **Snowflake pentamers**: in COLD AIHA, IgM autoantibodies form against RBC surface antigens, but only bind RBCs at low temperatures (below 30°C)
- 24. Snowflakes connecting red bottles: in cold AIHA, cold temperatures lead to agglutination of RBCs (due to cross-linking of IgM bound to RBC surfaces)
- 25. **Blue fingers**: COLD AIHA leads to blue, painful extremities (due to agglutination of RBCs)(extremities most susceptible because of relatively low temperature)
- 26. "HOT" food: WARM autoimmune hemolytic anemia (AIHA)
- 27. **Antibody meat stick**: In WARM AIHA, IgG autoantibodies form against RBC surface antigens
- 28. "Complimentary" coffee: in COLD and WARM AIHA, the complement system becomes activated (occurs because of surface immunoglobulins bound to cell surface)
- 29. "hOP ON IN": in COLD and WARM AIHA, immunoglobulin and complement act as OPSONINS (facilitate phagocytosis by macrophages)
- 30. **Meat bite**: AIHA (WARM > COLD) can lead to bite cells (due to macrophages removing pieces of complement-coated cell membrane)
- 31. **Round red tanks**: WARM and COLD AIHA lead to formation of spherocytes (pieces of cell membrane removed by macrophages → less membrane remaining → spherocytes → EXTRAVASCULAR hemolysis in spleen)
- 32. **Appetizers lysed on plate**: WARM and COLD AIHA can lead to INTRAvascular hemolysis (RBCs coated with complement → formation of membrane attack complexes [MAC] → hemolysis in blood vessels)
- 33. COON-skin cap: Coombs reagent (used in DIRECT Coombs test)
- 34. **Antibody sleeve holding antibody meat stick**: the Coombs reagent is an antibody that binds to an existing antibody on the RBC cell surface (used in DIRECT Coombs test, positive when reagent causes coated cells to agglutinate)
- 35. Blowing nose: in children, viral infections can cause WARM AIHA
- 36. Wolf: systemic lupus erythematosus (and other autoimmune diseases) can cause warm AIHA
- 37. Purple pencil: penicillin can cause warm AIHA
- 38. **myCOLDplasma showshoer** : Mycoplasma pneumoniae can cause COLD AIHA
- 39. Epstein's bar: epstein barr virus (EBV) can cause COLD AIHA
- 40. Chess board with B-archers and T-knights: lymphoma (particularly chronic lymphocytic leukemia CLL) can cause COLD AIHA

Blood & Coagulation 2.4 - Sickle Cell Disease



- 1. Sickle boats: sickle-shaped red blood cells (sickle cell anemia)
- 2. Iron sickle: hemoglobin S (HbS)
- 3. **Beta chariot with hemoglobin wheel**: Hb S is a result of a missense mutation in the BETA globin gene (on chromosome 11)
- 4. **Death VEIL**: in sickle cells, the GAG to GTC mutation in the Beta globin gene replaces GLUTAMIC ACID with VALINE
- 5. **Scared of water**: in sickle cells, hydrophobic interactions between hemoglobin molecules are increased (due to replacement of NEGATIVELY charged glutamic acid with NONPOLAR valine)
- 6. Clumped sickles: in sickle cells, the mutated hemoglobin molecules overlap and form polymers (due to increased hydrophobic interactions)
- 7. Crescent boats : sickle cells (polymerization of deoxy-HbS \rightarrow elongated rope-like fibers \rightarrow form fascicles that distort rbc)
- 8. Blue horseman : hypoxia promotes HgS polymerization and sickling (decreased O2 \rightarrow increased levels of DEOXY-HbS [60% threshold] \rightarrow increased polymerization)
- 9. **Dehydrated horseman** : cellular dehydration promotes HgS polymerization and sickling (rbc dehydration \rightarrow increased intracellular concentration of HbS \rightarrow increased polymerization)
- 10. **Acid horseman** : acidosis promotes HgS polymerization and sickling (acidity \rightarrow Hg dissociation curve shifts RIGHT \rightarrow Hg gives up oxygen more easily \rightarrow increased DEOXY-HbS \rightarrow increased polymerization)
- 11. Damaged dehydrating boat : sickle cells can be irreversibly damaged after multiple polymerization-depolymerization cycles \rightarrow persistent sickle shape \rightarrow potassium leakage \rightarrow cellular dehydration \rightarrow increased HbS concentration \rightarrow further polymerization and sickling)
- 12. Cracked stiffened hull : persistently sickled cells become stiff \rightarrow unable to alter their shape
- 13. **Red skulls in splenic cage**: sickled cells undergo EXTRAVASCULAR hemolysis in spleen (stiff sickled cells unable to alter shape → phagocytosis by macrophages in reticuloendothelial system)
- 14. Freely destroyed red skulls: sickled cells can undergo INTRAVASCULAR hemolysis (less common than extravascular)
- 15. Shy kid: sickle cell disease is autosomal recessive
- 16. **Single sickle & Beta sword**: sickle cell TRAIT occurs when only one beta globin allele is mutated (one HgS gene, one normal beta globin gene))
- 17. **Round boat**: in sickle cell TRAIT, the low concentration of deoxy-HbS produces enough normal beta subunits to avoid polymerization and sickling of rbcs (NO anemia)

- 18. **Strenuously paddling**: in sickle cell TRAIT, significant sickling can occur under EXTREME circumstances (severe hypoxia, severe dehydration (strenuous exercise), severe acidosis [sepsis])
- 19. **Double sickle**: two HbS mutations will result in sickle cell DISEASE (no normal beta chains)(causes anemia)
- 20. **Tethered boats**: sickle cells adhere to each other (due to development of extra adhesion molecules)(leads to vasoocclusion)
- 21. Big splenic shield : in sickle cell disease, the spleen becomes congested in early childhood (due to sequestration of sickled RBCs) \rightarrow splenomegaly
- 22. **Fatigued splenic soldier**: in sickle cell disease, the spleen becomes enlarged and dysfunctional in early childhood ("splenic fatigue")(due to occlusion of microvasculature by sickled RBCs → ischemia and fibrosis)
- 23. **Purple DNA collar**: in sickle cell disease, the peripheral blood smear displays Howell Jolly bodies (purple dots of old DNA in RBC cytosol)due to splenic fatigue (spleen normally removes old DNA from RBCs)
- 24. **Torn spleen hole**: in sickle cell disease, autosplenectomy occurs by adulthood (severely dysfunctional small fibrotic spleen)
- 25. $\mbox{\bf Pencil}$: in sickle cell disease, penicillin prophylaxis is recommended in early childhood
- 26. **Syringe**: in sickle cell disease, vaccination for encapsulated bacteria in childhood is critical (Strep pneumoniae, Neisseria meningitidis, Haemophilus influenzae)
- 27. **Sequestered into river** : sickle cells can cause splenic sequestration (large blockage of splenic circulation \rightarrow spleen rapidly swells with blood)(affects children)
- 28. **Sinking hemoglobin wheels**: splenic sequestration causes a precipitous drop in hemoglobin and hematocrit (due to spleen capturing RBCs)
- 29. **Fainting**: splenic sequestration causes hypotension (due to decreased intravascular volume)
- 30. **PLASTIC bone**: sickle disease can lead to APLASTIC crisis (infection with Parvovirus B19 → decreased production of RBCs by bone marrow → rapid decrease in hemoglobin and hematocrit [very problematic in sickle cell disease due to short lifespan of RBCs])
- 31. **Reticular tea bags floating to surface**: reticulocyte count is HIGH in splenic sequestration (bone marrow attempting to compensate) and LOW in aplastic crisis (low Hg production)

Blood & Coagulation 2.4 - Sickle Cell Disease



- 33. **Screaming skeleton**: sickle cell disease causes pain in various organs due to vasoocclusion (sickle cells get stuck in small capillaries)
- 34. **Transforming hand bones**: hands: sickle cell disease can cause dactylitis (swelling and pain in the fingers and toes ["hand-foot syndrome"] due to vasoocclusion in bone microvasculature)
- 35. **Bone deformity**: sickle cell disease can cause permanent necrosis and deformity of hands and feet (due to repeat episodes of dactylitis)
- 36. **Blackened femur head**: sickle cell disease can cause avascular necrosis of the femoral head (due to vasoocclusion and marrow expansion)
- 37. **Stabbed in the chest**: sickle cell disease can cause acute chest syndrome (vasoocclusion in lungs, causes severe pain and respiratory distress)(triggered by increased levels of deoxy-HbS in lungs [pulmonary infection, hypoventilation, atelectasis, thrombosis])
- 38. Skull and X bones: X-ray
- 39. **Fog obscuring pulmonary sails**: on chest X-ray, acute chest syndrome displays pulmonary infiltrates (resembles pneumonia)
- 40. Cracked helmet with black hair : sickle cell disease can cause ischemic stroke (due to vasoocclusion of cranial vessels)
- 41. **Pelvic sword**: sickle cell disease can cause priapism (sustained erection due to vasoocclusion, may cause ischemia and infarction)
- 42. **Boat leaking red**: sickle cell disease can cause hematuria (due to vasoocclusion and small infarcts in renal capillaries)
- 43. **Beat-up flank armour**: sickle cell disease can cause chronic kidney disease (due to repeated micro-infarcts in renal vasculature)
- 44. **Chipmunk face helmet**: sickle cell disease causes extramedullary hematopoiesis (chronic hemolysis → upregulation of RBC production → hematopoiesis expands out of medullary bone and into cortical bone [including cheek bones])
- 45. **Mohawk**: in sickle cell disease, extramedullary hematopoiesis into the skull causes a "crew cut" appearance on X-ray

- 46. **Mosquito netting**: sickle cell trait has a protective effect against Plasmodium falciparum
- 47. "A" flag: in sickle cell TRAIT, hemoglobin electrophoresis displays primarily a HbA1 band (normal adult hemoglobin, composed of alpha and beta subunits), and a smaller HgS band
- 48. "S" flag: in sickle cell DISEASE, hemoglobin electrophoresis displays primarily HbS (no HbA1 due to absence of normal beta globin)
- 49. **Holding baby**: in sickle cell DISEASE, fetal hemoglobin (HbF, gamma and alpha globin) is produced into adulthood (normally only produced until 6 weeks of age)
- 50. **Hydrotherapy area**; hydroxyurea is an important treatment for sickle cell DISEASE :
- 51. **Lifting baby**: hydroxyurea stimulates the production of fetal hemoglobin (HbF)(activates expression of gamma globin gene)
- 52. **Beta CHALICE**: sickle cell TRAIT (one copy of HbS mutation) in combination with another beta globin defect (i.e. beta thalassemia trait) results in a phenotype similar to sickle cell DISEASE
- 53. **C-shaped pike**: hemoglobin C (missense mutation in BETA globin gene replaces glutamic acid (negative charge) with lysine [positive charge] in position 6)(autosomal recessive)(homozygote has mild hemolytic anemia)
- 54. **Sickle plus "C" pike**: hemoglobin SC disease (one HbS mutation and one HbC mutation) results in a phenotype similar to sickle cell DISEASE (slightly milder)

Blood & Coagulation 2.5 - Megaloblastic Anemia: Folate Deficiency & Vitamin B12 Deficiency



- 1. "Fight of the Century": MACROcytic anemia (MCV > 100)
- 2. **MEGA BLAST pyrotechnics**: MEGALOBLASTIC anemia (a type of macrocytic anemia)(contains megaloblasts [large nucleated RBCs])
- 3. **Blasting RNA & lagging DNA**: MEGALOBLASTIC anemia is a result of DNA/RNA asynchrony (DNA maturation and cell division is delayed, while RNA translation and protein production continues)
- 4. Failure of fan division : in MEGALOBLASTIC anemia, cell DIVISION is impaired due to poor DNA maturation (meanwhile, cytoplasm continues to grow \rightarrow increased MCV)]
- 5. Packed crowd behind bone fence : in MEGALOBLASTIC anemia, the bone marrow is HYPERcellular
- 6. **Popped red balloon** : in MEGALOBLASTIC anemia, abnormal RBCs undergo apoptosis → few RBCs in circulation
- 7. Fan in macroCAGE : in MEGALOBLASTIC anemia, abnormal RBCs are phagocytosed by macrophages \rightarrow few RBCs in circulation
- 8. Falling purple tea bag: in MEGALOBLASTIC anemia, reticulocyte count is LOW (as a result of bone marrow dysfunction)(in contrast to hemolytic anemia, where reticulocyte count IS elevated)
- 9. **Oval purple firework** : megaloblasts are large oval cells with abundant basophilic cytoplasm
- 10. Firework with dark lacy center: the megaloblast nucleus contains lacy chromatin
- 11. First responders with multilobed pom-poms: MEGALOBLASTIC anemia is associated with HYPERsegmented neutrophils (five or more lobes)(due to abnormal cell maturation)
- 12. $\pmb{\text{Large firework}}$: MACROCYTIC MEGALOBLASTIC anemia can be caused by deficiency of folate or vitamin B12
- 13. IceBer9: vitamin B9 (folate)
- 14. "LEAFY": plants (particularly dark green leafy vegetables) and animal products contain folate
- 15. Counterclockwise costume rack: folate cycle
- 16. Double leaf: Dihydrofolate is produced from folate
- 17. Four leaf clover: TETRAhydrofolate is produced from Dihydrofolate (via the enzyme dihydrofolate reductase)
- 18. **Mask with "C" design** : Carbon groups (methyl groups) are added to TETRAhydrofolate
- 19. Flying purine shirt: folate is required for PURINE synthesis
- 20. Flying pyrimidine shirt: folate is required for PYRIMIDINE synthesis

- 21. **Discarded green leaves**: lack of consumption of leafy green vegetables can cause folate deficiency
- 22. "Fight Month": liver stores of folate can be depleted in just a few months
- 23. **Sponsored by goat's milk**: consumption of ONLY goat's milk can cause folate deficiency in infants (due to minimal folate in goat's milk)
- 24. **Broken pink rope**: insufficient small bowel surface area (i.e. celiac disease, short gut syndrome) can cause folate deficiency (because folate is absorbed in the duodenum and jejunum
- 25. **Drinking alcohol with liver cooler**: excessive alcohol consumption can cause folate deficiency (causes impaired uptake and storage of folate by the liver)
- 26. Cracked red bell: hemolysis
- 27. Chronic grandfather clock : chronic hemolysis can cause folate deficiency (persistently high RBC turnover \rightarrow rapid cell division and DNA synthesis \rightarrow depletion of folate stores
- 28. **Protruding red tongue**: glossitis (sore, swollen tongue) is a symptom of folate and B12 deficiency (due to rapidly dividing cells in the mouth)
- 29. "MTHFR": the enzyme MTHFR (methyl-tetrahydrofolate reductase) removes one carbon from TETRAhydrofolate
- 30. **Homing beacon**: homocysteine is converted into methionine using a carbon from TETRAhydrofolate (requires MTHFR activity)
- 31. "Mia's THe oNE": methionine is synthesized from homocysteine
- 32. Casting aside four-leaf clover jersey: folate is recycled back to the cycle once methyl groups have been removed from TETRAhydrofolate
- 33. "Home of the Sisters": homocysteine accumulates in the absence of available folate (due to inability to convert homocysteine to methionine

Blood & Coagulation 2.5 - Megaloblastic Anemia: Folate Deficiency & Vitamin B12 Deficiency

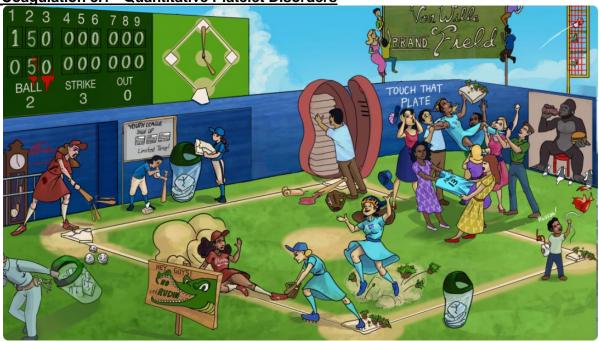


- 34. **Cobalt blue firework**: vitamin B12 (cobalamin)(a co-enzyme)(aids in methyltransferase reactions, including conversion of homocysteine to methionine)
- 35. "MMA": MethylMalonic Acid is converted to succinyl CoA by methylmalonyl CoA mutase (requires vitamin B12)
- 36. "SUXS": SUCCinyl CoA is produced from MMA
- 37. "1000": the liver holds a 1,000 day supply of vitamin B12 (takes a long time to deplete stores)
- 38. Faulty cobalt fireworks : vitamin B12 deficiency (causes macrocytic MEGALOBLASTIC anemia)
- 39. **Vegetable cape**: a vegan diet (complete absence of all animal products) can cause vitamin B12 deficiency (because B12 is not present in plant products)
- 40. **Alcoholic**: chronic alcohol consumption can cause vitamin B12 deficiency)
- 41. **Loss of lemons**: achlorhydria (lack of gastric acid production) can cause vitamin B12 deficiency (lack of acidic environment in the stomach → decreased pepsin → B12 is not cleaved from food)
- 42. "**No Intrinsic Right"**: decreased intrinsic factor can cause vitamin B12 deficiency (B12 needs to bind intrinsic factor in order to be absorbed)
- 43. **Destroying with antibodies**: pernicious anemia can cause vitamin B12 deficiency (autoantibodies destroy intrinsic factor and parietal cells in the stomach → lack of intrinsic factor → decreased vitamin B12 absorption
- 44. **Dirtying pancreas sponge**: pancreatic insufficiency can cause vitamin B12 deficiency (poor pancreas function \rightarrow trypsin does not cleave B12 from protein carriers in \rightarrow B12 unable to bind intrinsic factor)
- 45. Fiery pink rope : Crohn's disease can cause vitamin B12 deficiency (Crohn's disease injures the ileum \to poor absorption of B12 from the ileum)

- 46. **Wormy rope**: the tapeworm Diphyllobothrium latum can cause vitamin B12 deficiency (by competing for B12 in the ileum)
- 47. **Discarding myelin sweatbands**: vitamin B12 deficiency causes decreased myelination (B12 is important for myelin synthesis)(causes neuropsychiatric impairment)
- 48. "Super Combined Tournament": vitamin B12 deficiency causes Subacute Combined Degeneration (injury to both the dorsal and lateral columns due to demyelination)
- 49. **Pyramid**: vitamin B12 deficiency causes degeneration of MOTOR pathways in the LATERAL PYRAMIDAL TRACTS (causes weakness, paralysis)
- 50. **Column**: vitamin B12 deficiency causes degeneration of the SENSORY pathways in the DORSAL SPINAL COLUMN)(causes paresthesias, numbness, sensory ataxia)
- 51. **Toppling leafy greens**: avoid treating vitamin B12 deficiency with folate (corrects the anemia, but can worsen neurological symptoms
- 52. **Chair sticks**: high serum homocysteine levels are associated with a hypercoagulable state (including cardiovascular, cerebrovascular disease)

Blood & Coagulation 3.1 - Quantitative Platelet Disorders





- 1. "150" count : thrombocytopenia is defined as a platelet count less than 150,000 per microliter
- 2. **Red "50" tiles**: bleeding risk with thrombocytopenia typically doesn't increase until platelets fall to 50,000 per microliter (SPONTANEOUS bleeding may occur at under 20,000 per microliter)
- 3. Falling plate clock: quantitative platelet disorders (thrombocytopenia) cause an increase in bleeding time (no effect on PT/aPTT)
- 4. **Split antibody bat on shattered plate** : immune thrombocytopenia (ITP) is a group of disorders caused by IgG autoantibodies against glycoprotein 2b/3a receptors on platelets→ thrombocytopenia
- 5. **Spleen shaped plate receptacle**: IgG coated platelets in ITP are phagocytosed by macrophages in the spleen
- 6. Sneezy ITP little leaguer: ITP in children is thought to be caused by abnormal autoimmune response to viral infection and typically affects children from 2-5 years old (slight male predominance)
- 7. Home platelet bleeding: PLATELET bleeding is characterized by petechiae, epistaxis, and easy bruising
- 8. 3 month youth league: ITP in children is usually self-limited, lasts 3-6 months, and typically only requires conservative treatment
- 9. **Grandfather clock by home plate**: ITP in adults has many potential causes (both infectious and autoimmune) and has a CHRONIC course with episodic acute flares
- 10. Young woman with \lg bat : in adults, ITP typically affects young women in their 20-30's
- 11. moon facies: treatment of ITP includes immunosuppression with corticosteroids]
- 12. IVIG keys: in severe cases, IVIG may be used to treat acute ITP flares
- 13. **Dragging spleen trash can away** : splenectomy is used to treat refractory cases of ITP (splenectomy stops phagocytosis of platelets by splenic macrophages→ increased platelet counts)
- 14. **Heparin hunter getting HIT**: heparin-induced thrombocytopenia (HIT) is an autoimmune destruction of platelets seen after administration of heparin (mostly LMWH)
- 15. Player wearing "4" glove touching plate with antibody studs: HIT is caused by development of autoantibodies that bind the platelet factor 4-heparin complex and also attach directly to platelets via their Fc region
- 16. **Platelet player degranulating with joy** : antibody binding of platelets in HIT causes them to degranulate→ release of cytokines, platelet factor 4, and ADP→ clot formation
- 17. **HITting dirt clods**: platelet activation in HIT leads to platelet aggregation and clot formation→ DVT, pulmonary embolism (arterial thrombosis occurs less frequently)
- 18. **Dirt clods covering plate**: systemic thrombosis and platelet consumption are one mechanism leading to thrombocytopenia in HIT
- 19. **Splenic trash can by first base**: the major cause of thrombocytopenia in HIT is phagocytosis of IgG coated platelets in the spleen
- 20. Flame bandana: patients with HIT often have subtle clinical findings such as fever, chills, tachycardia, or dyspnea
- 21. "**No intRUDIN**" **GATOR**: treatment of HIT consists of ANTIcoagulation with direct thrombin inhibitors such as bivalRUDIN or arGATROban

- 22. "**Touch That Plate**": thrombotic thrombocytopenic purpura (TTP) a thrombotic microangiopathy)
- 23. "HUSsah!": hemolytic-uremic syndrome (HUS) a thrombotic microangiopathy
- 24. **Diffuse gathering spectators touching plate**: the thrombotic microangiopathies (TTP/HUS) cause DIFFUSE platelet activation—thrombocytopenia
- 25. Clods covering up plate : thrombocytopenia in TTP-HUS is caused by diffuse thrombus formation→ platelet consumption→ thrombocytopenia
- 26. Clods and sheared ball in foul pole mesh: thrombosis of the microcirculation increases shear force on erythrocytes→ hemolysis→ microangiopathic hemolytic anemia (MAHA)
- 27. **E Cola**: over 90% of cases of HUS are caused by infection with enterohemorrhagic E. coli (EHEC)
- 28. **Red stool**: children with HUS can present with symptoms of EHEC infection (e.g. bloody diarrhea, abdominal pain, vomiting)
- 29. **Hamburger**: children with HUS can present with symptoms of EHEC infection (transmitted in undercooked meat/ unpasteurized dairy)
- 30. ${\bf She-gorilla}: {\bf Shiga-like}$ toxin produced by EHEC is responsible for development of HUS
- 31. **Tears in inner poster** : Shiga-like toxin (from EHEC) induces capillary endothelial damage→ inflammation and thrombosis→ MAHA + thrombocytopenia (HUS)
- 32. Hussah! with covered plate, popped balloon, and twisty straw: the triad of findings in HUS includes thrombocytopenia, microangiopathic hemolytic anemia, and acute kidney injury (most common cause of severe AKI in children <5)
- 33. Young woman Touching That Plate : thrombotic thrombocytopenic purpura (TTP) is most commonly seen in young women
- 34. **Torn ADAMS #13 jersey between antibody dresses**: TTP (acquired) involves development of autoantibodies directed against the ADAMTS13 protease
- 35. **Crowded Von Wille BRAND sign** : TTP is caused by a deficiency in ADAMTS13 (normally cleaves large vWF complexes) → large vWF complexes activate/cross-link platelets→ intravascular thrombosis
- 36. Covered plate, popped balloon, and twisty straw: HUS triad (thrombocytopenia, hemolytic anemia, AKI) is also seen in TTP (+fever, neuro sx)
- 37. Flame hat on TTP player: fever is part of the TTP pentad (+thrombocytopenia, HA, AKI, neuro sx)
- 38. **Brain hat falling off**: neurologic symptoms (ranging from confusion to seizures) caused by microvascular injury are part of TTP pentad (+thrombocytopenia, HA, AKI, fever)
- 39. **Stuffing splenic locker**: splenomegaly (e.g. portal hypertension causes venous congestion of the splenic vein) increases sequestration of platelets in the spleen → thrombocytopenia)
- 40. **Broken bat marrow**: marrow suppression (drugs, infection, cancer, etc.) can cause thrombocytopeni

Blood & Coagulation 3.2 - Qualitative Platelet Disorders



- 1. Bleeding clock: in QUALITATIVE platelet dysfunction, BLEEDING TIME is prolonged (platelet numbers, PT, and aPTT are normal)
- 2. Bruises, red spots, and nosebleed : platelet DYSFUNCTION causes "platelet" type bleeding (skin and mucous membranes bleed → easy bruising, petechiae, epistaxis)
- 3. Elevated BUN bag: uremia (due to kidney disease) can cause QUALITATIVE platelet dysfunction
- 4. "Jurriasic park": platelet dysfunction due to uremia (uremic toxins interfere with platelet aggregation and adhesion → "platelet-type" bleeding)
- 5. Cast & neck brace: trauma can cause QUALITATIVE platelet dysfunction (lactic acidosis and temperature dysregulation → impaired platelet aggregation and adhesion - "platelet-type" bleeding)
- 6. Holding up diaSWEETES: diabetes mellitus can cause QUALITATIVE platelet dysfunction (leads to THROMBOTIC complications [rather than
- 7. Fibrous dirt CLODs: thrombus formation is a complication of diabetes mellitus (chronic hyperglycemia → dysregulation of signaling pathways → HYPERactive platelets → increased thrombosis)
- 8. Broken guitar string: myocardial infarction is a complication of diabetes mellitus (chronic hyperglycemia \rightarrow HYPERactive platelets \rightarrow increased thrombosis [includes MI])
- 9. St BERNARD: Bernard-Soulier syndrome (an inherited QUALITATIVE platelet disorder)(caused by deficiency of GP1b receptors on platelet surface)
- 10. Hiding kid: Bernard-Soulier syndrome is autosomal recessive
- 11. "16" jersey: glycoprotein 1b (GP1b) receptors on platelets are responsible for adhering platelets to von Willebrand factor (vWf) → eventually leads to formation of platelet plug
- 12. Unable to adhere to "von Wille Brand Field" wall: in Bernard-Soulier syndrome (absence of GP1b on platelets), platelets are unable to adhere to vWf \rightarrow no formation of platelet plug \rightarrow excessive bleeding
- 13. Giant satellite dish: in Bernard-Soulier syndrome, giant platelets are visible in the peripheral blood (GP1b is involved in process of platelet budding from megakaryocytes → absence of GP1b leads to formation of large platelets)
- 14. Unsuccessful WRIST aggregation: in Bernard-Soulier syndrome, the ristocetin test is NEGATIVE (normally causes vWf to bind to GP 1b) (deficient GP1b → decreased binding to vWf → decreased platelet aggregation)

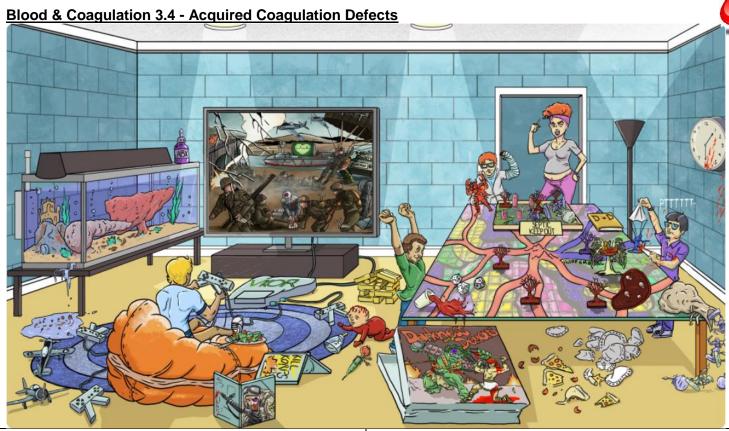
- 15. No improvement with falling red drink: in a ristocetin test for Bernard-Soulier syndrome, addition of normal serum to the unaggregated sample does NOT lead to platelet aggregation (because the normal serum does NOT contain GP1b)
- 16. GT car hood: Glanzmann Thrombasthenia (an inherited QUALITATIVE platelet disorder)
- 17. Receding kid: Glanzmann thrombasthenia is autosomal recessive
- 18. 2 balls & 3 strikes: in Glanzmann thrombasthenia, glycoprotein IIb/IIIa (GP2a/3b, located on platelet surface) is dysfunctional
- 19. Unable to reach fibrous branch: in Glanzmann's thrombasthenia, fibrinogen is unable to bind to the dysfunctional GPIIb/IIIa \rightarrow reduced formation of platelet plug → "platelet-like" bleeding pattern
- 20. Successful WRIST grab: in Glanzmann thrombasthenia, the ristocetin test is POSITIVE (i.e. normal)(because vWF and Gp1b are both normal in this disorder)
- 21. WASp: Wiskott-Aldrich Syndrome (an inherited QUANTITATIVE and QUALITATIVE platelet disorder)
- 22. Hiding behind an "X": Wiskott-Aldrich syndrome is X-linked recessive
- 23. Wiskott wasps: Wiskott-Aldrich syndrome is caused by mutations in the 'Wiskott-Aldrich Syndrome' protein (WASP) (a cytoskeletal protein in platelets, B cells, T cells)
- 24. Red wasp stings: Wiskott-Aldrich syndrome causes eczema
- 25. Fallen broken plates: Wiskott-Aldrich syndrome causes a QUANTITATIVE platelet deficiency (i.e. thrombocytopenia)
- 26. Small beat-up plates: Wiskott-Aldrich syndrome causes a QUALITATIVE platelet deficiency -> small, deformed platelets
- 27. Plates in MacroCAGE; in Wiskott Aldrich syndrome, small deformed platelets are phagocytosed by splenic macrophages contributes to thrombocytopenia:
- 28. Crutches: Wiskott-Aldrich syndrome causes immunodeficiency (a combined T cell and B cell deficiency)

Blood & Coagulation 3.3 - Inherited Coagulation Defects



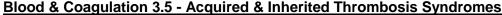
- 1. **Von Wille Brand**: von Willebrand disease (the most common inherited coagulopathy)
- 2. **Torn "Von Wille Brand"**: von Willebrand disease is due to a quantitative deficiency of von Willebrand Factor
- 3. Epithelial tiles: squamous endothelium lining arteries and veins
- 4. Dominoes: Von Willebrand disease is autosomal dominant
- 5. **broken endothelial tile**: von Willebrand factor is exposed with endothelial injury (located in the SUB-endothelium)
- 6. Paddle on pucks: von Willebrand factor is responsible for aggregation of platelets (by linking glycoprotein 1b [on platelet surface] to exposed collagen [on endothelium])
- 7. **Bloody clock**: von Willebrand disease causes prolonged bleeding time (due to decreased platelet function)
- 8. **Bloody clock**: von Willebrand disease causes prolonged bleeding time (due to decreased platelet function)
- 9. Falling "8" ball : in von Willebrand disease, factor 8 is decreased (decreased von Willebrand factor \rightarrow factor 8 is more susceptible to degradation \rightarrow shorter factor 8 half-life)
- 10. **Long "PTTTT" sound**: von Willebrand disease leads to prolonged aPTT (decreased factor 8 levels → decreased activity of INTRINSIC coagulation pathway)
- 11. **Bruising & nose bleed**: von Willebrand disease leads to mild "platelet" like bleeding (easy bruising, epistaxis, prolonged skin bleeding, heavy menstrual bleeding)
- 12. Losing red bag items: von Willebrand disease causes heavy menstrual bleeding
- 13. **WRIST bracelet**: the ristocetin test (ristocetin is added to blood sample → ristocetin activates GP1b receptors on platelets → GP1b receptors bind to von Willebrand factor multimers → platelet aggregation)
- 14. **Unsuccessful WRIST aggregation**: in von Willebrand disease, the ristocetin test is NEGATIVE (decreased von Willebrand factor → decreased binding to GP1b receptors → decreased platelet aggregation)
- 15. **Red drink falling on aggregated pucks**: in ristocetin test for von Willebrand disease, normal blood (with normal amount of von Willebrand factor) can be added to a sample with unaggregated platelets (subsequent platelet aggregation confirms von Willebrand disease)
- 16. **DESert-mobile**: desmopressin (DDAVP)(a synthetic analog of vasopressin) is used to treat von Willebrand disease
- 17. **Von Wille Brand flag**: desmopressin causes endothelial cells to release stored von Willebrand factor
- 18. "CRYO" pack : cryoprecipitate treats severe hemorrhage from von Willebrand disease

- Stenotic princess hat: aortic stenosis can cause acquired von Willebrand disease (shearing effect from narrowed aortic valve → destruction of von Willebrand factor multimers)
- 20. **Tangled red strings**: angiodysplasia combined with von Willebrand disease causes intractable gastrointestinal bleeding
- 21. "A" pool rack : hemophilia A (an inherited coagulopathy)
- 22. Falling "8" ball : hemophilia A is due to a deficiency of factor 8
- 23. **Hiding behind crossed pool cues** : hemophilia A is an X-linked recessive disorder
- 24. Falling "10" ball: in hemophilia A, deficiency of factor 8 disrupts the intrinsic coagulation pathway as well as the the common pathway (because of decreased activation of factor 10)
- 25. **Bloody open knee**: hemophilia A leads to hemarthroses (bleeding in joints)("coagulopathic" type bleeding; also includes intramuscular bleeding, intracranial hemorrhage)
- 26. "8" ball root canal : in hemophilia A, dental procedures can cause hemorrhage
- 27. "PTTTT": hemophilia A leads to prolonged aPTT (due to disruption of the the INTRINSIC pathway)
- 28. **DESert-mobile**: desmopressin is used to treat hemophilia A (induces platelets and endothelial cells to release factor 8)
- 29. "B"illiards: hemophilia B (an inherited coagulopathy)
- 30. **Fallen "9" ball**: hemophilia B is due to deficiency of factor 9 (X-linked recessive; affects INTRINSIC pathway; prolonged aPTT; "coagulopathic" bleeding)
- 31. **Wolf attacking "8" ball** : systemic lupus erythematosus can be associated with anti-factor 8 antibodies → increased clearance of factor 8 → "acquired hemophilia"
- 32. **Pregnant woman**: pregnancy and postpartum can cause anti-factor 8 antibodies \rightarrow increased clearance of factor 8 \rightarrow "acquired hemophilia"
- 33. Rheumatic lanterns : rheumatoid arthritis can be associated with antifactor 8 antibodies \rightarrow increased clearance of factor 8 \rightarrow "acquired hemophilia"
- 34. **Purple pencil**: penicillin can cause anti-factor 8 antibodies \rightarrow increased clearance of factor 8 \rightarrow "acquired hemophilia"
- 35. **Broken "MIX" tape** : a mixing study can be used to diagnose the presence of anti-factor 8 antibodies (normal serum is added to patient's serum \rightarrow antibodies inactivate factor 8 \rightarrow aPPT does NOT correct)
- 36. **Moon face**: corticosteroids can treat acquired hemophilia from antifactor 8 antibodies



- 1. **DICe** : disseminated intravascular coagulation (DIC) (an acquired coagulopathy)
- 2. **Thrombotic trees**: DIC leads to excessive clotting (due to activation of the coagulation system)
- 3. **Hemorrhaging red drink**: DIC leads to excessive bleeding (due to activation of the fibrinolytic system)
- 4. "SEPTIC": sepsis can cause DIC
- 5. **Pink gravestones**: gram-negative sepsis (Neisseria meningitidis, etc) is the most common cause of DIC (bacterial proteins and lipopolysaccharides activate coagulation)
- 6. Thrombotic trees : in DIC, exposure to procoagulant substances leads to activation of the coagulation cascade $\,$
- 7. **Cancer crab** : malignancy (especially acute promyelocytic leukemia) can cause DIC
- 8. Cast: trauma can cause DIC
- 9. Pregnant mom: obstetric complications can cause DIC
- 10. Losing coagulation fighters: DIC causes depletion of both pro- and anti-coagulant components (due to excessive activation of coagulation cascade)("CONSUMPTIVE COAGULOPATHY")
- 11. Red arm spots & bleeding from spear entry points: DIC causes "platelet" type bleeding (petechiae, ecchymoses, mucocutaneous bleeding, oozing from IVs or arterial lines)
- 12. $\hbox{\bf Head bleeding}: \hbox{DIC causes "coagulopathy" type bleeding (GI, genitourinary, or cerebral hemorrhages)}$
- 13. Clot in "ILIOFEMORAL" river and lung tree: DIC can cause venous thrombi (most commonly DVTs in iliofemoral veins +/- pulmonary emboli)
- 14. **Red skull lagoon**: DIC can cause tissue and organ infarction (due to arterial thrombi)
- 15. **ParaTrooper**: DIC leads to prolonged PT (due to consumption of coagulation factors in EXTRINSIC pathway)
- 16. "PTTTT": DIC leads to prolonged aPTT (due to consumption of coagulation factors in INTRINSIC pathway)
- 17. **Bloody clock**: DIC leads to prolonged bleeding time (due to thrombocytopenia from platelet consumption)
- 18. Half-eaten pepperoni : DIC causes formation of schistocytes (RBCs squeeze through small arteries full of fibrin clots \rightarrow mechanical damage to RBC membrane)
- 19. Raised D&D guide: DIC causes elevated D-dimer (a fibrin degradation product)
- 20. Empty "Kill Korps" case : Vitamin K deficiency (an acquired coagulopathy)

- 21. **Brigades 2, 7, 9, 10** : factors 2, 7, 9, 10 (vitamin K-dependent factors)
- 22. "C" captain & "S" sergeant : protein C and S (vitamin K-dependent factors)
- 23. **Colonic chair**: Vitamin K is synthesized by colonic bacteria (from precursors in green leafy vegetables, olive and soybean)
- 24. Liver VKOR console powering K controllers: Vitamin K is activated by the liver enzyme epoxide reductase (VKOR)
- 25. **Gamma bandages**: Vitamin K is a cofactor for gamma-glutamyl carboxylase (activates Vitamin K-dependent factors)
- 26. **Baby fumbling with K controller**: newborns are at increased risk of Vitamin K deficiency (partially due to GI tract lacking intestinal flora that synthesize Vitamin K; partially due to decreased utilization of Vitamin K by the liver)
- 27. **Broken breast-shaped bottle**: breast milk contains very little Vitamin K (contributes to increased risk of Vitamin K deficiency in newborns)
- 28. **Red hair streaks**: Vitamin K deficiency in infants presents with hemorrhagic disease of the newborn (intracranial hemorrhage, retroperitoneal bleeding, death)
- 29. Syringe rattle toy : newborns are routinely given a Vitamin K injection to decrease the risk of Vitamin K deficiency
- 30. **House blocks**: home birth is a risk factor for Vitamin K deficiency in newborns (due to absence of Vitamin K injection)
- 31. **leaking debris** : diseases that cause malabsorption (inflammatory bowel disease, chronic pancreatitis, etc) can cause Vitamin K deficiency
- 32. **Fish antibiotics** : antibiotics can cause Vitamin K deficiency (by killing GI bacteria that synthesize Vitamin K)
- 33. **Liver rock**: cirrhosis can cause Vitamin K deficiency (by impairing Vitamin K activation by epoxide reductase in the liver)
- 34. **Red stream out of gun & bloody nose**: in adults, Vitamin K deficiency causes "coagulopathic" type of bleeding (if mild: easy bruising, epistaxis, prolonged bleeding after injury) (if severe: GI and genitourinary bleeding)
- 35. ParaTrooper : Vitamin K causes prolonged PT (reduced factor $7 \rightarrow$ decreased activation of EXTRINSIC pathway)
- 36. FFP fighter jets: Vitamin K deficiency can be treated with fresh frozen plasma (FFP)(contains all Vitamin K-dependent factors)
- 37. **"K" controller** : mild Vitamin K deficiency can be treated with Vitamin K supplementation







- 1. **Numerous piles of sticks** : acquired and inherited thrombosis syndromes
- 2. Phospholipid barbed wire with antibody supports:
 ANTIPHOSPHOLIPID SYNDROME (an ACQUIRED thrombosis disorder; causes clots in both venous and arterial systems) (a result of autoantibodies against cardiolipin and other phospholipids/proteins)
- 3. **Inflamed bone lantern**: antiphospholipid syndrome can be secondary to rheumatoid arthritis
- 4. White wizard hat: antiphospholipid syndrome can be secondary to HIV
- 5. **Lupus wolf** : antiphospholipid syndrome can be secondary to systemic lupus erythematosus
- 6. **Arrow to the HEART**: antiCARDIOlipin antibodies are common in antiphospholipid syndrome
- 7. **Telescope for spiral galaxy screening**: RPR and VDRL (screening tests for syphilis] may be falsely positive if anticardiolipin antibodies are present
- 8. Arrows in wolf tree: LUPUS ANTICOAGULANT antibodies (INHIBIT clotting in laboratory, PROMOTE clotting in situ) are common in antiphospholipid syndrome
- 9. **"PTTTT"**: in antiphospholipid syndrome, the aPTT may be prolonged due to the presence of LUPUS ANTICOAGULANT
- 10. Arrows in glycoprotein vines: ANTI-BETA2-GLYCOPROTEIN antibodies are common in antiphospholipid syndrome
- 11. **Thrombotic sticks in iliofemoral river** : antiphospholipid syndrome presents with VENOUS thromboses in a young patient (e.g. DVT)
- 12. **Nest clot in pulmonary tree** : antiphospholipid syndrome may present with pulmonary embolism
- 13. **Black-striped helmet**: antiphospholipid syndrome also presents ARTERIAL thromboses in a young patient (e.g. TIA or ischemic stroke)
- 14. **Broken guitar string**: antiphospholipid syndrome also presents ARTERIAL thromboses in a young patient (e.g. in coronary arteries causing myocardial infarction)
- 15. Flaming heart lanterns; antiphospholipid syndrome can present with Non-Bacterial Thrombotic Endocarditis (thrombosis \rightarrow damage to valve endothelium \rightarrow activation of complement and platelets \rightarrow valve vegetations :
- 16. **Woman clutching baby**: antiphospholipid syndrome can present with pregnancy-related complications (miscarriages, severe preeclampsia) (microthrombi in placenta impair nutrient and oxygen delivery to fetus)
- 17. **Empty baby carriage**: antiphospholipid syndrome can present with recurrent miscarriages
- 18. Fallen cobalt blue fireworks: vitamin B12 (cobalamin) deficiency (similar to B6 and B9 deficiency) can cause an ACQUIRED thrombosis syndrome)

- 19. **BLASTing blue firework**: B12 deficiency causes megaloBLASTic anemia (due to decreased DNA production)
- 20. **Raised HOMing beacon**: HOMOcysteine levels are elevated in vitamin B6, B9, and B12 deficiency (because these vitamins are involved in homocysteine metabolism) (elevated homocysteine levels increase thrombosis risk)
- 21. **Stuck in mud**: hyperviscosity syndromes increase thrombosis risk (higher viscosity → increased resistance → stasis → thrombosis)
- 22. Excessive lily pads: polycythemia (increased RBCs) can cause thrombosis (due to hyperviscosity syndrome)
- 23. Excessive antibody twigs: high levels of immunoglobulins in the blood (e.g. multiple myeloma, Waldenstrom's macroglobulinemia) can cause thrombosis (due to hyperviscosity syndrome)
- 24. **Raising sickle**: sickle cell disease can cause thrombosis (rigid sickle-shaped RBCs cause stasis in small vessels → hyperviscosity syndrome)
- 25. **Cracked helmet**: hyperviscosity syndrome can present with neurologic symptoms and confusion (due to stasis in cerebral vessels)
- 26. Falling muddy glasses : hyperviscosity syndrome can present with blurred vision (due to stasis in ocular vessels)
- 27. Laid factor "V" bear trap : factor V Leiden (most common INHERITED thrombosis syndrome)
- 28. Young Caucasian stuck in "V" bear trap : factor V Leiden occurs most commonly in younger Caucasians
- 29. **Domino wall**: inherited thrombosis syndromes are usually autosomal dominant (e.g. factor V Leiden, prothrombin G20210A mutation, Protein C/S deficiency, ATIII deficiency)
- 30. Captain unable to deactivate "V" trap: in factor V Leiden, protein C is unable to destroy activated factor V (because factor V Leiden mutation eliminates the binding spot for protein C)
- 31. **Dam over-built by THROM-beaver** : in factor V Leiden, thrombin activity is increased (increased factor V activity \rightarrow increased generation of thrombin AND loss of negative feedback loop \rightarrow increased risk of VENOUS thrombosis)
- 32. **Thrombotic sticks in iliofemoral river**: most inherited thrombosis syndromes (e.g. factor V Leiden, prothrombin G20210A mutation, Protein C/S deficiency, ATIII deficiency) present with VENOUS thromboses (e.g. DVT, PE)
- 33. **OCP canteen**: in factor V Leiden, combined oral contraceptives and pregnancy increase the risk of thrombosis
- 34. Smoker: in factor V Leiden, smoking increases the risk of thrombosis
- 35. **Immobilized leg**: in factor V Leiden, prolonged immobilization increases the risk of thrombosis



Blood & Coagulation 3.5 - Acquired & Inherited Thrombosis Syndromes



- 36. **THROM-beaver**: thrombin (catalyzes the conversion for fibrinogen to fibrin)
- 37. **Baby THROM-beaver**: PRO-thrombin (precursor to thrombin) (mutated in PROTHROMBIN G20210A MUTATION [an INHERITED thrombosis syndrome])
- 38. **Domino wall**: inherited thrombosis syndromes are usually autosomal dominant (e.g. factor V Leiden, prothrombin G20210A mutation, Protein C/S deficiency, ATIII deficiency)
- 39. Camp "G20210A": G20210A is the mutation in PROTHROMBIN GENE (guanine replaced by adenine)
- 40. Family of baby THROM-beavers : PROTHROMBIN G20210A MUTATION leads to overproduction of prothrombin \rightarrow increased risk of VENOUS thrombosis
- 41. **Thrombotic sticks in iliofemoral river**: most inherited thrombosis syndromes (e.g. factor V Leiden, prothrombin G20210A mutation, Protein C/S deficiency, ATIII deficiency) present with VENOUS thromboses (e.g. DVT, PE)
- 42. **Hiding Captain**: protein C deficiency (an INHERITED thrombosis syndrome)
- 43. **Hiding Sergeant**: protein S deficiency (an INHERITED thrombosis syndrome)
- 44. **Domino wall**: inherited thrombosis syndromes are usually autosomal dominant (e.g. factor V Leiden, prothrombin G20210A mutation, Protein C/S deficiency, ATIII deficiency)
- 45. **Holding up 5 fingers** : protein C or S deficiency → increased factor Va activity → increased risk of VENOUS thrombosis
- 46. Raising "8" binoculars : protein C or S deficiency \rightarrow increased factor VIIIa activity \rightarrow increased risk of VENOUS thrombosis
- 47. **Thrombotic sticks in iliofemoral river**: most inherited thrombosis syndromes (e.g. factor V Leiden, prothrombin G20210A mutation, Protein C/S deficiency, ATIII deficiency) present with VENOUS thromboses (e.g. DVT, PE)
- 48. "No WAR": in protein C and S deficiency, avoid treatment with WARFARIN (protein C and S are vitamin-K dependent) (administration of warfarin leads to further decreased protein C and S activity → increased thrombotic effect)
- 49. **Black splotches**: in protein C and S deficiency, administration of warfarin can cause warfarin-induced skin necrosis (small thrombi in skin lead to necrosis and extravasation in tissues → purpura and skin necrosis)
- 50. **Doll with black splotches**: in newborns, homozygous protein C or S deficiency can cause neonatal purpura fulminans (complete lack of protein C or S \rightarrow severe arterial and venous thromboses, DIC, necrotic hemorrhagic skin lesions)

- 51. **THROM-beaver cage** : antithrombin III (ATIII) (normally has an inhibitory effect on thrombin, factors IX and X)
- 52. Broken THROM-beaver cage with escaped beaver : ATIII DEFICIENCY (an INHERITED thrombosis syndrome) (leads to decreased inhibition of thrombin \rightarrow increased thrombin activity \rightarrow increased risk of VENOUS thrombosis) (normal PT, aPTT, and bleeding time)
- 53. **Domino wall**: inherited thrombosis syndromes are usually autosomal dominant (e.g. factor V Leiden, prothrombin G20210A mutation, Protein C/S deficiency, ATIII deficiency)
- 54. **Ineffective Hunter**: in ATIII DEFICIENCY, heparin has no effect (does not prolong aPTT) (because heparin must bind antithrombin in order to function) (normally heparin inhibits factor II, IX, X [INTRINSIC pathway])
- 55. **Dumping protein from kidney bucket** : nephrotic syndrome can cause an ACQUIRED ATIII DEFICIENCY (loss of antithrombin in the urine → increased thrombosis risk)
- 56. **Thrombotic sticks in iliofemoral river**: most inherited thrombosis syndromes (e.g. factor V Leiden, prothrombin G20210A mutation, Protein C/S deficiency, ATIII deficiency) present with VENOUS thromboses (e.g. DVT, PE)
- 57. "No intrudin" & big gater: direct thrombin inhibitors ("rudin" and "gatran" suffixes) are used to treat antithrombin deficiency (because heparin is not effective in the absence of functioning antithrombin)

Myeloid Disorders 1.1 - Myeloproliferative Neoplasms & Myelodysplastic Syndromes





- 1. **Spots of red, blue, pink, and white** : the MYELOID cell line includes RBCs; granulocytes (basophils, eosinophils, neutrophils); monocytes (\rightarrow macrophages, dendritic cells); mast cells; and megakaryocytes (\rightarrow platelets)
- 2. Blasting immature blue, white & pink splotches: ACUTE myeloid leukemia (AML) is caused by proliferation of immature granulocyte and monocyte precursors (myeloBLASTS) in the bone marrow
- 3. Mile-Pro truck delivering mature art: myeloproliferative neoplasms (overproduction of mature myeloid cell types) include chronic myelogenous leukemia, polycythemia vera, essential thrombocythemia, and primary myelofibrosis
- 4. **Mature white, pink & blue spots**: CHRONIC myelogenous leukemia (CML) is caused by the proliferation of more mature GRANULOCYTE progenitor cells (eosinophil, neutrophil, basophil precursors) in bone marrow
- 5. "BReakABLE": CML always arises from a stem cell that has acquired the BCR-ABL fusion gene
- 6. "PHILADELPHIA": Philadelphia chromosome (truncated chromosome 22 containing BCR-ABL fusion gene)
- 7. **TIRE swing**: the BCR-ABL fusion protein is a constitutively activated receptor TYROSINE kinase
- 8. **Independently active tire jack**: constitutively active JAK2 mutation (cytoplasmic tyrosine kinase) causes polycythemia vera, essential thrombocythemia, and primary myelofibrosis
- 9. Abundant red lilies: polycythemia vera (a myeloproliferative neoplasm with increase in hemoglobin, hematocrit, and RBC count)
- 10. **Abundant plates from bone path**: essential thrombocythemia (a myeloproliferative neoplasm with increased number of platelets)
- 11. **Plates and saucers**: essential thrombocythemia is associated with platelets of variable size
- 12. **Hyperlobulated table on bone path**: essential thrombocythemia is associated with increased number of megakaryocytes (enlarged and hyperlobulated) in the bone marrow
- 13. **Pile of toothpicks**: essential thrombocythemia increases risk of thrombotic events (MI, DVT, PE)
- 14. **Bloody toothpick** : essential thrombocythemia increases risk of bleeding → ecchymosis, epistaxis, menorrhagia, gingival bleeding
- 15. **Unzipped Von Wille brand**: Willebrand factor may be deficient (used up by platelets) in essential thrombocythemia)
- 16. **ASA Umpire**: Aspirin can be used to decrease thrombotic complications in essential thrombocythemia
- 17. **Bone trees**: primary myelofibrosis (a myeloproliferative neoplasm characterized by bone marrow fibrosis)
- 18. Filled with pink, blue, and white flowers : primary myelofibrosis begins with a hyperproliferative phase (hypercellular bone marrow with increased GRANULOCYTES and megakaryocytes)
- 19. **Multiple megakaryocyte platters**: early in primary myelofibrosis, the bone marrow may show increased numbers of MEGAKARYOCYTES)

- 20. **Irregular drink spill**: early in primary myelofibrosis, the bone marrow shows megakaryocyte atypia (irregular nuclei with coarse chromatin)
- 21. Shark watering can for bone tree : in primary myelofibrosis, abnormal megakaryocytes stimulate fibroblast growth \rightarrow collagen deposition \rightarrow bone marrow fibrosis
- 22. **Dry bone cup** : in primary myelofibrosis, bone marrow aspiration results in a "dry tap"
- 23. **Red liver and spleen statue**: primary myelofibrosis can cause hepatosplenomegaly (due to extramedullary hematopoiesis)
- 24. **Refusing food**: massive splenomegaly (due to extramedullary hematopoiesis) can cause early satiety
- 25. **Black marked spleen**: massive splenomegaly (due to extramedullary hematopoiesis) can lead to splenic infarcts
- 26. Pale dress: primary myelofibrosis can cause anemia
- 27. **Variable purple and white**: primary myelofibrosis is associated with variable leukocyte and platelet counts
- 28. **Different red spheres**: primary myelofibrosis is associated with nucleated RBCs of varying size (anisocytosis) and shape (poikilocytosis)
- $29.\,\text{Red tear}$: primary myelofibrosis is associated with teardrop shaped RBCs (dacrocytes)
- 30. **Immature pink, white, and blue spots**: primary myelofibrosis is associated with granulocyte precursors (myelocytes, metamyelocytes, and bands) in the periphery
- 31. **Sweaty flame bandana**: myeloproliferative neoplasms cause severe constitutional symptoms (fever, night sweats, and weight loss, and fatigue)
- 32. **Hydro rock area**: hydroxyurea can be useful in the treatment of myeloproliferative neoplasms (reduces platelets)
- 33. **Rock pen nib**: ruxolitinib is a JAK2 inhibitor can be useful in the treatment of myeloproliferative neoplasms (especially polycythemia vera and myelofibrosis)
- 34. **Dirty disorganized bone**: the myelodysplastic syndromes (dysplastic and ineffective myeloid cell production)
- 35. Old man: the myelodysplastic syndromes are most common in the elderly
- 36. **Different sized plastic flowers**: in the myelodysplastic syndromes, the bone marrow is hypercellular (with dysplastic erythrocyte and granulocyte precursors)
- 37. **Empty pan** : the myelodysplastic syndromes cause pancytopenia (despite bone marrow hypercellularity) \rightarrow weakness, bleeding, infections
- 38. **Blue ring around hat**: the myelodysplastic syndromes are associated with ringed sideroblasts on peripheral smear (premature RBCs with iron deposits in mitochondria as a ring around the nucleus)
- 39. **Pointing to immature paint splotches**: the myeloproliferative neoplasms and myelodysplastic syndromes can all transform into AML (blast phase transformation)
- 40. **Central fibrotic bone tree**: the myeloproliferative neoplasms and myelodysplastic syndromes can all cause myelofibrosis (fibrotic phase)

Myeloid Disorders 1.2 - Acute Myeloid Leukemia (AML) & Chronic Myeloid Leukemia (CML)



- 1. **Bloody red crab next to bone**: leukemia (starts in the bone marrow) (neoplastic cells spill into the peripheral blood)
- 2. Immature blue, white & pink splotches: ACUTE myeloid leukemia (AML) is caused by proliferation of immature granulocyte and monocyte precursors (myeloblasts) in the bone marrow (ACUTE = rapid disease onset and progression)
- 3. **BLASTing blue, white & pink paint**: myeloBLASTS (immature cells of the MYELOID lineage) are precursors to granulocytes (basophil, neutrophil, eosinophils) and monocytes)
- 4. **Mature white, pink & blue spots**: CHRONIC myelogenous leukemia (CML) is caused by the proliferation of more mature GRANULOCYTE progenitor cells (eosinophil, neutrophil, basophil precursors) in bone marrow
- 5. **Grandfather clock**: Most patients with CML present in the CHRONIC phase, with insidious onset of symptoms
- 6. **Grey-haired abstract artist**: AML primarily affects older adults (average age 65 years)
- 7. **Broken crab chemistry set**: exposure to chemotherapy (i.e. history of cancer treatment) is a risk factor for AML
- 8. Radiation symbol fan : exposure to ionizing radiation is a risk factor for AML
- 9. **Benzene ring design**: exposure to benzene (plastics, rubber tire industries) is a risk factor for AML
- 10. "DOWNtown": Down syndrome is a risk factor for AML
- 11. "**Museum of Myeloid Art**": myeloproliferative disorders (polycythemia vera, essential thrombocythemia, primary myelofibrosis, CML) & myelodysplastic syndrome can transform into AML
- 12. **Empty pan**: AML presents with functional pancytopenia (thrombocytopenia, anemia, functional neutropenia [despite leukocytosis])
- 13. White dove accumulation: AML may present with LEUKOCYTOSIS due to proliferation of myeloblasts (despite a reduced number of functioning WBCs)
- 14. Sleeping artist: AML presents with weakness and fatigue (secondary to anemia)
- 15. Flame bandana: AML presents with fever (due to infection)
- 16. **Bony pants**: AML presents with bone pain (due bone marrow expansion)
- 17. **Red dots & purple splotches**: AML presents with petechiae and ecchymosis (due to thrombocytopenia)

- 18. "20%" satellite dish: in AML, myeloblasts account for a minimum of 20% of bone marrow cells or 20% of peripheral WBC cells
- 19. **Granular sundial with large irregular center**: myeloblasts contain abundant basophilic cytoplasm (with faint granules); and large folded or bilobed nuclei (with multiple dark nucleoli)
- 20. **Pink rods on HOURly sundial**: in some AML subtypes, AUER rods (pink rod-shaped structures) are present in the cytoplasm of myeloblasts)
- 21. **Hydrogen peroxide paint remover** : Auer rods stain positive for peroxidase
- 22. "GO PRO": acute PROmyelocytic leukemia (APL) (characterized by PROmyelocytes [immature myeloid cells with abundant Auer rods])
- 23. 15yo translocating PRO jacket onto 17yo RA-RA cheerleader: in APL, a chromosome 15-17 translocation creates a PML/RARalpha fusion protein (an abnormal retinoic acid receptor; stops myeloid differentiation at the PROmyelocytic phase)
- 24. **DICe**: APL can lead to disseminated intravascular coagulation (DIC) (due to secretion of tissue factor by promyelocytes)
- 25. **Embroidered "A"**: APL can be treated with all-trans-retinoic acid (a Vitamin A analog) (signals neoplastic promyelocytes to differentiate into mature neutrophils)

Myeloid Disorders 1.2 - Acute Myeloid Leukemia (AML) & Chronic Myeloid Leukemia (CML)



- 26. **Grey-haired pointillism artist**: CML primarily affects older adults (average age 60 years)
- 27. Radiation symbol fan : exposure to ionizing radiation is a risk factor for CML
- 28. "BReakABLE": CML always arises from a stem cell that has acquired the BCR-ABL fusion gene (due to translocation of the ABL gene [chromosome 9] adjacent to the BCR gene [chromosome 22)])
- 29. "PHILADELPHIA": Philadelphia chromosome (truncated chromosome 22 created by a balanced reciprocal translocation between chromosome 9 and 22) (contains BCR-ABL fusion gene)
- 30. **TIRE swing**: the BCR-ABL fusion protein is a constitutively activated receptor tyrosine kinase
- 31. White dove accumulation: CML presents with leukocytosis (due to abundant granulocytes [both MATURE and PARTIALLY MATURE forms])
- 32. "Pro Gym", "Myelo Mall" & "Metal Co" : CML presents with IMMATURE granulocytes (e.g. in sequence of development : myeloBLASTS (rare) → PROmyelocytes → MYELOcytes → METAmyelocytes)
- 33. **Tall "Myelo Mall"**: in CML, MYELOcytes are present in greater numbers than other myeloid precursors ("MYELOcytic bulge")
- 34. Abundant platelet paint palettes : CML presents with thrombocytosis
- 35. **Thrombotic sticks**: CML can present with thrombosis (due to excessive platelets)
- 36. **Empty "Von Wille Brand" cup** : in CML, excessive platelets can consume available von Willebrand factor → bleeding
- 37. **Red paint**: CML can present with hemorrhage (due to depletion of von Willebrand factor by excessive platelets)
- 38. White smock: CML presents with anemia (normochromic, normocytic)
- 39. Large spleen statue: CML can cause splenomegaly (due to extramedullary hematopoiesis)
- 40. **Spleen skull**: CML can present with splenic infarcts (due to insufficient perfusion of enlarged spleen) (presents with LUQ pain)
- 41. **Progressively faster, more abstract paint splotches**: in CML, granulocytes may become more immature over time (as a result of progression from the chronic stage to a more aggressive "accelerated stage")

- 42. **Broken palettes**: the CML "accelerated stage" presents with thrombocytopenia
- 43. **BLASTing spray paint**: CML may progress to "BLAST crisis" (rapid production of myeloBLASTs [appears similar to AML])
- 44. **Pen "NIBs"**: CML can be treated with tyrosine kinase inhibitors (imatiNIB, dasatiNIB, nilotiNIB) (inhibit the constitutive tyrosine kinase activity of BCR-ABL)
- 45. **Massive white dove accumulation**: LEUKEMOID reaction (WBC count > 50,000) (BENIGN)
- 46. **First responders**: in LEUKEMOID reaction, the excessive WBCs are primarily NEUTROPHILs
- 47. **Bacterial lanterns**: LEUKEMOID reaction is often secondary to infection
- 48. **BANDages**: in LEUKEMOID reaction, BANDs (slightly immature neutrophils) are present in peripheral blood
- 49. **Weight shifting LEFT**: LEUKEMOID reaction causes a LEFT shift in the WBC distribution (because of BANDS in the peripheral blood)
- 50. **Raising white chalk**: in LEUKEMOID reaction, leukocytes alkaline phosphatase (LAP) is NORMAL or ELEVATED (due to presence of functional neutrophils) (in contrast, LAP is LOW in CML due to dysfunctional neutrophils)
- 51. **GRANULAR bird poops**: during an INFECTIOUS or INFLAMMATORY process, NEUTROPHILS display toxic granulation, Dohle bodies [peripheral light blue spots of RER], and cytoplasmic vacuoles (seen in LEUKEMOID reaction, NOT in CML)

Myeloid Disorders 1.3 - Polycythemia





- 1. Abundant red lilies: polycythemia (increase in hemoglobin, hematocrit, and RBC count)
- 2. **Elevated hemoglobin wheels**: hemoglobin and hematocrit are increased in polycythemia
- 3. Counting bunch of red flowers : RBC count is increased in polycythemia
- 4. Red flowers per "dL": hemoglobin, hematocrit, and RBC count are CONCENTRATIONS (affected by changes in plasma volume)
- 5. **Dehydrated red lilies**: RELATIVE polycythemia (increased hemoglobin, hematocrit, and RBC count secondary to LOW PLASMA VOLUME)(most common polycythemia)
- Capped red EPO paint: in RELATIVE polycythemia, erythropoietin (EPO) levels are normal (normal O2 levels → no physiologic need for more RBCs])
- 7. "MASS" scale with total body rose pattern: RBC MASS is the total MASS of RBCs in the body (not affected by plasma volume)
- 8. Increased red flower MASS : ABSOLUTE polycythemia (increased production of RBCs → increased hemoglobin, hematocrit, RBC count, and RBC MASS)
- 9. Painting red lilies while lifting one finger: PRIMARY polycythemia
- 10. BONE easel : PRIMARY polycythemia is due to an abnormal BONE marrow process → production of excessive number of RBCs
- 11. **Vera** : polycythemia VERA (a myeloproliferative disorder that causes PRIMARY polycythemia)
- 12. **Pink kidney palette** : in polycythemia VERA, excess RBC production leads to an well oxygenated kidney)
- 13. **Discarded red EPO paint**: in polycythemia vera, EPO production is DOWNREGULATED (excessive RBCs → more than adequate oxygenation of kidney → kidney decreases EPO production)
- 14. **Independently active tire jack**: polycythemia VERA is caused by mutations in the JAK2 gene (cytoplasmic tyrosine kinase)(mutation leads to constitutive activation of STAT pathway → uncontrolled hematopoietic cell proliferation)(most commonly V617F)
- 15. **Tall plate stack** : polycythemia VERA causes proliferation of platelets (thrombocytosis)
- 16. **Pink, blue, and white paint tubes**: polycythemia VERA causes proliferation of granulocytes (neutrophils, basophils, eosinophils)
- 17. Embarrassed red art student : polycythemia VERA causes facial plethora (excessive RBCs → tissue congestion → ruddy complexion)
- 18. **Kidney & liver splotches**: polycythemia VERA causes hepatosplenomegaly (excessive RBCs → tissue congestion in liver and spleen)
- 19. Dark red sludge: polycythemia VERA increases blood viscosity
- 20. **Thrombotic sticks**: polycythemia VERA increases the risk of arterial and venous thrombosis (due to increased blood viscosity)

- 21. **Losing glasses with brain hat**: polycythemia VERA can cause headaches, dizziness, and vision changes (due to small foci of thrombosis and hemorrhage in the CNS)
- 22. **Clutching belly**: polycythemia VERA can cause abdominal pain (due to abnormal mucosal blood flow in the gastrointestinal system)
- 23. **Scratching from warm mosquito-ridden water** : polycythemia VERA can cause aquagenic pruritus (warm bath or shower → release of histamine from mast cells → pruritus)
- 24. **Yellow knitting needles**: polycythemia VERA can cause hyperuricemia (excessive RBCs → increased breakdown of RBC DNA → increased purine metabolism → increased uric acid)
- 25. **Yellow yarn ball on toe** : polycythemia VERA can cause gout (due to hyperuricemia)
- 26. **Fibrotic bone tree**: myelofibrosis is a late complication of polycythemia VERA (bone marrow becomes replaced by fibroblasts and collagen)
- 27. **Enlarged liver and spleen spots**: myelofibrosis secondary to polycythemia VERA can cause severe hepatosplenomegaly (due to extramedullary hematopoiesis)
- Abstract blasts of pink, blue, and white paint: polycythemia VERA
 can transform into acute myelogenous leukemia (AML)(results in
 proliferation of myeloblasts)
- 29. **TWO** raised red paint tubes: SECONDARY polycythemia (excessive RBC production as a result of INCREASED EPO levels)
- 30. **Blue face**: PHYSIOLOGIC SECONDARY polycythemia is driven by hypoxia
- 31. **Blue kidney palette**: in PHYSIOLOGIC SECONDARY polycythemia, kidney hypoxia → increased production of EPO by the kidney → stimulates proliferation of RBC precursors in the bone marrow)
- 32. Blue bloater & pink puffer: COPD can cause PHYSIOLOGIC SECONDARY polycythemia (as a result of chronic hypoxia)
- 33. **Obese snorer with blue face**: obstructive sleep apnea can cause PHYSIOLOGIC SECONDARY polycythemia (as a result of chronic hypoxia)
- 34. **Blue heart design**: cyanotic congenital heart disease can cause PHYSIOLOGIC SECONDARY polycythemia (as a result of chronic hypoxia)
- 35. **Blue face standing up high**: high altitude can cause PHYSIOLOGIC SECONDARY polycythemia (low atmospheric O2 → chronic hypoxia)
- 36. **Red paint tubes caught in net**: INAPPROPRIATE SECONDARY polycythemia (a result of EPO production outside the kidney [usually secondary to tumors])
- 37. Crabs in kidney net : renal cell carcinoma is the most common cause of INAPPROPRIATE SECONDARY polycythemia
- 38. **Happy pink face**: O2 saturation is normal in INAPPROPRIATE SECONDARY polycythemia (because EPO is produced independent of hypoxia)

Myeloid Disorders 2.1 - Acute Lymphoblastic Leukemia (ALL), Chronic Lymphocytic Leukemia (CLL), Hairy Cell Leukemia & Adult T-cell Leukemia



- Bloody bone marrow crab: lymphoid neoplasms (e.g. ALL/CLL) can present as LEUKEMIA (neoplastic lymphoid cells arise in the bone marrow → cells leak into peripheral circulation)
- 2. **Chess board**: lymphoid neoplasms (e.g. ALL/CLL) can present as LYMPHOMA (neoplastic lymphoid cells form a tumor in a lymphoid organ [lymph node, spleen, etc] OUTSIDE of the bone marrow)
- 3. Immature lymphoid archers and knights: ACUTE lymphoBLASTIC leukemia/lymphoma (ALL) is caused by proliferation of immature B and T-lymphocyte precursors (lymphoBLASTS) in bone marrow/lymphoid tissue (ACUTE = rapid disease onset and progression)
- 4. Young child with #1 : ALL is the most common cancer in children (peak incidence 2-5 years of age)
- 5. "Downtown": Down syndrome is a risk factor for ALL
- 6. Immature archer with bone quiver: B-cell ALL is caused by a mutation in a pre-B cell in the bone marrow (most common ALL type)
- 7. Immature knight in THYME garden: T-cell ALL is caused by a mutation in a pre-T-cell in the THYMUS (site of T-cell maturation)
- 8. **Teenaged knight**: T-cell ALL usually occurs in male teenagers (age 15-20)
- 9. **Blast attack**: ALL (both B- and T-cell) presents with lymphoBLASTs (early stage of lymphocyte development) on the peripheral smear
- 10. **Purple shield with thin border**: LYMPHOblasts contain a large nucleus and scant cytoplasm
- 11. **PASSing frisbee**: LYMPHOblasts are periodic acid-Schiff (PAS) POSITIVE (due to presence of glycogen in the cytoplasm) (MYELOblasts are negative)
- 12. "TdT" weapon rack: LYMPHOblasts are terminal deoxynucleotidyl transferase (TdT) POSITIVE (MYELOblasts are negative) (TdT is required for VDJ recombination in developing lymphocytes)
- 13. 2,3,4,5,7,8 footwork: pre-T LYMPHOblasts express CD2, CD3, CD4, CD5, CD7, CD8
- 14. 10, 19, 20 archery target: pre-B LYMPHOblasts express CD10, CD19, CD20
- 15. "25" gate: a diagnosis of acute lymphoblastic LEUKEMIA requires > 25% blasts in the bone marrow (+/- a lymphoid mass) (common manifestation of B-cell ALL)
- 16. **Blasting knights off chessboard**: acute lymphoblastic LYMPHOMA is diagnosed if < 25% blasts in the bone marrow & presence of lymphoid mass) (common manifestation of T-cell ALL)
- 17. **Dummy chest sack**: T-cell ALL (leukemia or lymphoma) often presents with a large anterior mediastinal mass

- 18. **Node necklace**: T-cell ALL (leukemia or lymphoma) often presents with lymphadenopathy (particularly cervical, supraclavicular, axillary)
- Squeezing neck: an anterior mediastinal mass can present with superior vena cava syndrome (due to SVC compression), stridor or dyspnea (due to tracheal compression), or dysphagia (due to esophageal compression)
- 20. **Empty pan**: ALL presents with functional pancytopenia (anemia, thrombocytopenia) (neutropenia despite leukocytosis) due to proliferation of lymphoblasts in bone marrow
- 21. **Bloody apron**: ALL presents with easy bruising or bleeding (due to thrombocytopenia)
- 22. Flame bandana: ALL presents with fever (due to neutropenia)
- 23. White dove accumulation: ALL presents with leukocytosis (due to abundant lymphoblasts in peripheral blood)
- 24. **Hepatic oat bag & splenic saddle**: ALL presents with hepatosplenomegaly (due to infiltration by leukemic cells)
- 25. **Lymph node ornaments**: ALL presents with lymphadenopathy (due to infiltration by leukemic cells)
- 26. Painful kick by bony leg: ALL can cause bone pain (due to bone marrow expansion and infiltration of periosteum by leukemic cells)
- 27. **Brain helmet**: ALL tends to metastasize to the central nervous system (meningeal involvement can causes headaches)
- 28. **Testicular tassels**: ALL tends to metastasize to the testicles (causes testicular mass)
- 29. Wise old T knight: ADULT T-CELL LEUKEMIA (presents in older adults)
- 30. "T" capsid helmet: ADULT T-CELL LEUKEMIA is caused by infection with human T-lymphotropic virus type 1 (HTLV-1)(a retrovirus)
- 31. **Helper squire with "4" belt**: HTLV-1 infection causes uncontrolled proliferation of mature CD4+ helper T cells (therefore ADULT T-CELL LEUKEMIA is CD4 positive)
- 32. **Horse kicking over TdT rack**: ADULT T-CELL LEUKEMIA is negative for TdT (because TdT is a lymphoBLAST marker)
- 33. **Red skin marks**: ADULT T-CELL LEUKEMIA can present with red plaques and papules (due to skin infiltration by leukemic cells) (appears similar to cutaneous T-cell lymphoma)
- 34. **Bony leg**: ADULT T-CELL LEUKEMIA can cause lytic bone lesions (due to bone infiltration)
- 35. Raising milk bottle: ADULT T-CELL LEUKEMIA can cause hypercalcemia (due to lytic bone lesions)

Myeloid Disorders 2.1 - Acute Lymphoblastic Leukemia (ALL), Chronic Lymphocytic Leukemia (CLL), Hairy Cell Leukemia & Adult T-cell Leukemia



- 36. **Mature archers on old bone tapestry**: chronic lymphocytic leukemia (CLL) is caused by abnormal proliferation of mature B lymphocytes in bone marrow
- 37. Old king: CLL presents in older adults (average age 60 years)
- 38. **Grandfather clock**: CLL is a chronic disease (follows an indolent course)
- Bone army: in CLL, mutations in a mature B cell lead to proliferation of clones in the bone marrow
- 40. BraCLet with 2 archer charms: in CLL, neoplastic cells exhibit increased expression of the proto-oncogene BCL2 (inhibits apoptosis → prolongs cell survival)
- 41. "1" spear & "9" shield : in CLL, neoplastic cells express B-cell markers (CD19 and greater)
- 42. "5" bow : in CLL, neoplastic cells express CD5 (usually a T-cell marker!)
- 43. **Abundant B archers on periphery**: CLL presents with leukocytosis (primarily B-cells)
- 44. Ink SMUDGE: CLL presents with "SMUDGE cells" on peripheral smear (due to collapse of fragile tumor cells during slide preparation)
- 45. **Nodal necklace**: CLL may present with lymphadenopathy (due to infiltration with leukemic cells)
- 46. **Spleen & liver designs**: CLL may present with hepatosplenomegaly (due to infiltration with leukemic cells)
- 47. **Chessboard**: CLL can present as a LYMPHOMA ("SMALL LYMPHOCYTIC LYMPHOMA" [SLL])
- 48. **Rows of small B-archers**: SLL displays sheets of small B-cells within lymphoid organs
- 49. **Mutant B-archer taking out normal B-archer** : neoplastic B cells suppress the function of normal B cells
- 50. **Losing antibody arrows**: CLL presents with HYPOgammaglobulinemia (due to decreased production of immunoglobulins by abnormal B-cells)
- 51. **Bacterial tassels**: CLL presents with bacterial infections (due to HYPOgammaglobulinemia)
- 52. **Antibody toothpicks**: in CLL, abnormal B cells can produce autoantibodies → autoimmune destruction of RBCs and platelets → anemia and thrombocytopenia
- 53. **Empty pan**: CLL presents with functional pancytopenia (anemia and thrombocytopenia secondary to autoantibodies, splenic sequestration, and bone marrow crowding by neoplastic cells) (dysfunctional WBCs despite leukocytosis)

- 54. **Hairy bear on aging tapestry**: HAIRY CELL LEUKEMIA (a chronic lymphoid leukemia)
- 55. B-cell bear bow: HAIRY CELL LEUKEMIA is a B-cell neoplasm
- 56. **Middle aged hunter**: HAIRY CELL LEUKEMIA is most common in middle-aged men
- 57. **Bear TRAP**: HAIRY CELL LEUKEMIA stains positive for Tartrate-Resistant Acid Phosphatase (TRAP)
- 58. Large spleen sac: HAIRY CELL LEUKEMIA causes massive splenomegaly (due to infiltration of the reticuloendothelial system)
- 59. Fibrotic bone tree: HAIRY CELL LEUKEMIA presents with bone marrow fibrosis (due to infiltration by leukemic cells
- 60. **Empty pan**: HAIRY CELL LEUKEMIA presents with pancytopenia (due to bone marrow infiltration and fibrosis)
- 61. **Empty bone cup**: HAIRY CELL LEUKEMIA may present with a "dry tap" (bone marrow biopsy fails to produce contents) due to extensive myelofibrosis

Myeloid Disorders 2.2 - Hodgkin Lymphoma



- 1. **Chess board**: lymphoma (tumors that originate in the lymphoid tissue)(divided into Hodgkin and non-Hodgkin types)
- 2. $\textbf{B-Archers}: \mathsf{B}$ lymphocytes (the mutated cells in Hodgkin lymphoma)
- 3. **Owl nests**: germinal center (location of B cell somatic hypermutation [mutating to produce selective antibodies)] and class-switching [from IgM to other Ig antibodies]
- Hodgwig the owl's eggs in nest: Hodgkin lymphomas stem from a mutated B lymphocyte in the germinal center
- IDENTICAL owlettes: Hodgkin lymphoma manifests as proliferation of CLONAL B cells (due to mutation of a single B cell within the germinal center or shortly after leaving germinal center)
- Beaded necklace: Hodgkin lymphoma most commonly arises within germinal centers of LYMPH NODES
- 7. Liver rock : Hodgkin lymphoma can arise within germinal centers in the LIVER
- 8. **Spleen rock**: Hodgkin lymphoma can arise within germinal centers in the SPLEEN
- 9. **Stepwise owl perches**: Hodgkin lymphoma spreads to adjacent lymph nodes in a contiguous pattern
- 10. **Neck owl**: Hodgkin lymphoma commonly arises within the CERVICAL or SUPRACLAVICULAR lymph nodes
- 11. **Owl on chest MAST**: Hodgkin lymphoma commonly arises within the mediaSTINAL lymph nodes
- 12. **Owl eyes**: Hodgkin lymphoma ALWAYS displays Reed-Sternberg cells (cells with two nuclear lobes containing an acidophilic nucleolus surrounded by a clear zone)
- 13. **Blueish owl poops**: on histology, Hodgkin lymphoma displays a background of inflammatory cells (lymphocytes, eosinophils, neutrophils, fibroblasts, etc) with sparse (but always present!) Reed-Sternberg cells
- 14. "XXX" & "VVV" feathers : in Hodgkin lymphoma, Reed-Sternberg cells express CD15 and CD30
- 15. **Epstein's Beer**: Hodgkin lymphoma is associated with Epstein-Barr virus (EBV) infection
- 16. "NF Charms Book': Hodgkin lymphoma can be associated with activation of NF-kappaB (as a result of EBV infection)(helps mutant B-cells evade apoptosis)
- 17. **Forked wand**: a personal or family history of autoimmune disorders (rheumatoid arthritis, SLE, sarcoidosis, etc.) is a risk factor for Hodgkin lymphoma
- 18. **Bimodal rug**: Hodgkin lymphoma has a bimodal age distribution (peaks at age 20 and age 65)

- 19. **CytoCOINS**: in Hodgkin lymphoma, cytokines cause "B" (constitutional) symptoms
- Shrinking and sweaty: Hodgkin lymphoma presents with "B" symptoms (fever, night sweats, and unintentional weight loss)
- 21. #1: NODULAR SCLEROSIS is the most common type of Hodgkin lymphoma (particularly in developed countries)
- 22. **NESTS separated by FIBROUS bark**: NODULAR SCLEROSIS Hodgkin lymphoma displays NODULES of inflammatory cells separated by FIBROUS collagen
- 23. **Light beams**: NODULAR SCLEROSIS Hodgkin lymphoma displays LACUNAR Reed-Sternberg cells (retraction of cytoplasm during slide fixation → ring of clear space ["lacunae"] around cell nucleus)
- 24. **MIXING potions**: MIXED CELLULARITY Hodgkin lymphoma (2nd most common type)
- 25. **Globe design**: MIXED CELLULARITY Hodgkin lymphoma is the more common in developing countries
- 26. **Epstein's Beer**: MIXED CELLULARITY Hodgkin lymphoma is strongly associated with Epstein-Barr virus (EBV) infection
- 27. **White wizard**: HIV infection is a risk factor for MIXED CELLULARITY Hodgkin lymphoma (particularly in the setting of EBV co-infection)
- 28. **Belt buckle**: MIXED CELLULARITY Hodgkin lymphoma often arises in abdominal lymph nodes
- 29. **Spleen pocket**: MIXED CELLULARITY Hodgkin lymphoma often arises in the spleen
- 30. **Freely bubbling NODULES**: MIXED CELLULARITY Hodgkin lymphoma displays NODULES without fibrous septae
- 31. **Hodg-rid outcast**: LYMPHOCYTE PREDOMINANT ("non-classic") Hodgkin lymphoma
- 32. Absence of owl companion: LYMPHOCYTE PREDOMINANT Hodgkin lymphoma does NOT contain Reed-Sternberg cells (therefore no expression of CD15 and CD3)
- 33. **Puffy popcorn**: LYMPHOCYTE PREDOMINANT Hodgkin lymphoma displays LYMPHOHISTIOCYTIC cells (multilobed puffy nucleus)
- 34. "XX" & "XIX" stitching: in LYMPHOCYTE PREDOMINANT Hodgkin lymphoma, LYMPHOHISTIOCYTIC cells express CD19 and CD20
- 35. **African-American male**: LYMPHOCYTE PREDOMINANT Hodgkin lymphoma is most common in males of African descent

Myeloid Disorders 2.3 - Non-Hodgkin Lymphoma





- 1. Chess board with b-cell archers: lymphoma (tumors that originate in the lymphoid tissue)(divided into Hodgkin and non-Hodgkin types)
- 2. Crossed out Hodgwig owl: non-Hodgkin lymphoma (NHL)
- 3. Adult archer pieces: NHL is most commonly a result of proliferation of MATURE B lymphocytes
- 4. Bone box : the bone marrow is a PRIMARY lymphoid organ (where B-cells originate and begin the maturation process)
- 5. Archers heading into SPLEEN sac : immature B-cells migrate to the SPLEEN, where they gain the ability to make IgD (in addition to IgM)
- Archers heading to chessboard: mature B-cells migrate to SECONDARY lymphoid organs (spleen, lymph nodes, tonsils, specialized tissue in GI and respiratory tracts)
- 7. Shooting IgM arrows : some mature B-cells become IgM-producing plasma cells (T-cell independent) \rightarrow secrete IgM from the bone marrow
- Shooting IgG arrows: some mature B-cells (those that are activated by T-cells) become specialized plasma cells (secrete IgG, IgA, IgE from the bone marrow)
- 9. "XIX" & "XX" stitching: in non-Hodgkin lymphoma, the neoplastic cells (MATURE B-cells) express the B-cell markers CD19 and CD2
- 10. Crossed out owl's eyes: non-Hodgkin lymphoma (NHL) does not have Reed-Sternberg cells (unlike Hodgkin lymphoma)
- 11. **Sporadic bat nodes**: NHL usually affects multiple lymph nodes in a NON-contiguous distribution (unlike contiguous spread in Hodgkin lymphoma)
- 12. **Scattered chess pieces**: NHL commonly involves EXTRA-nodal secondary lymphoid tissues (spleen, tonsils, GI tract, respiratory tract) and NON-lymphoid tissues (CNS) (unlike Hodgkin lymphoma)
- 13. "Burkitt" sign: BURKITT lymphoma (an aggressive B-cell lymphoma)
- 14. "Translocating cabinet": Burkitt t(8;14), follicular t(14;18), marginal cell t(11;14), and extra marginal zone lymphoma t(11;18) are associated with specific gene translocations
- 15. "8" door above "14" candelabra : BURKITT lymphoma is caused by a translocation between chromosome 8 to 14 t(8;14)
- 16. MYC mice : in BURKITT lymphoma, the t(8;14) translocation leads to overexpression of the c-MYC oncogene
- 17. **MYC** mice growing in size : in BURKITT lymphoma, overexpression of the c-MYC oncogene leads to unregulated cell growth → RAPID tumor expansion (c-MYC codes for a growth-stimulating transcription factor)
- 18. Raising "Ki-67" broom: BURKITT lymphoma has a high Ki-67 fraction because of its rapid growth (Ki-67 is a protein present during cellular replication)]
- 19. **Dividing broom fibers**: BURKITT lymphoma displays abundant mitotic figures (cells in the process of dividing) (due to rapid cell division)
- 20. "Starry sky" cloak: BURKITT lymphoma displays a "starry sky" appearance (macrophages surrounded by clear spaces, interspaced within sheets of blue tumor cells)
- 21. "Africa" cauldron : ENDEMIC BURKITT lymphoma is most common in equatorial Africa and New Guinea)
- 22. Respirator mouthpiece : ENDEMIC BURKITT lymphoma presents as a large jaw tumor
- 23. **Kid shopper**: BURKITT lymphoma primarily occurs in children (in both ENDEMIC and NON-ENDEMIC forms)

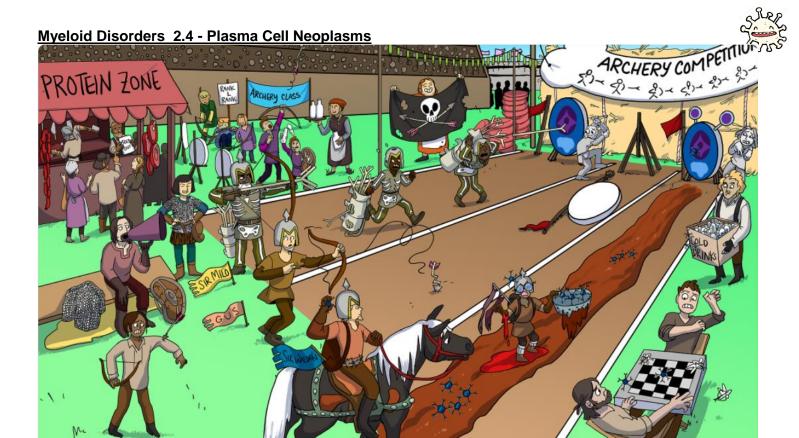
- 24. **Holding potion mid-belly**: NON-ENDEMIC BURKITT lymphoma presents with a rapidly growing ABDOMINAL mass (bowel, ovary, or retroperitoneal tissues)
- 25. Crutch: BURKITT lymphoma can be due to IMMUNODEFICIENCY
- 26. **Old store owner**: IMMUNODEFICIENCY-related BURKITT lymphoma occurs in older adults
- 27. Lymph node necklace: IMMUNODEFICIENCY-related BURKITT lymphoma presents with LYMPHADENOPATHY
- 28. **Wizard hat**: IMMUNODEFICIENCY-related BURKITT lymphoma usually occurs in HIV-infected patients
- 29. **Epstein's bar**: ENDEMIC and NON-ENDEMIC BURKITT lymphoma are associated with EBV infection
- 30. "Falling" sash: FOLLicular lymphoma (a NHL)
- 31. Sash from "14" candle to 18" fire tool : FOLLICULAR lymphoma is caused by a translocation between chromosome 14 and 18 t(14;18)
- 32. **Double "invincibility" BRACelet**: in FOLLICULAR lymphoma, the t(14;18) translocation results in overexpression of the BCL2 (B-cell lymphoma-2 gene) oncogene (inhibits apoptosis)(also in some DIFFUSE LARGE B-cell lymphomas)
- 33. **Nodular fire**: FOLLICULAR lymphoma displays round hypercellular nodules (appear similar to the FOLLICLES and germinal centers within normal lymph nodes) (originates from mutated B-cells in the GERMINAL CENTER
- 34. **Waxing and waning moon centerpiece**: FOLLICULAR lymphoma presents with generalized waxing and waning lymphadenopathy
- 35. **Transforming into DIFFUSELY LARGE patron**: FOLLICULAR lymphoma can transform into DIFFUSE LARGE B-cell lymphoma
- 36. LARGE antibody archer: DIFFUSE LARGE B-cell lymphoma (a NHL)
- 37. Pointing with "1" finger: DIFFUSE LARGE B-cell lymphoma is the most common lymphoma in adults
- 38. **Hexagon bracelet**: some DIFFUSE LARGE B-cell lymphomas display alterations (gene rearrangements, mutations in the promoter region) in the BCL-6 gene (regulates DNA transcription)
- 39. Large jars : DIFFUSE LARGE B-cell lymphoma displays sheets of large B cells
- 40. **Wizard drinking "Epstein's bar" beer**: EBV and HIV co-infection can cause DIFFUSE LARGE B-cell lymphoma)
- 41. White wizard hat : in HIV infected patients, lymphoma is an AIDS-defining condition
- 42. **HERMES statue with "8" staff**: HHV-8 and HIV co-infection can cause PRIMARY EFFUSION lymphoma (a NHL) (presents in the pleura, peritoneum, or pericardial spaces)
- 43. **Brain jar**: NHL can present as a primary CNS lymphoma (usually DIFFUSE LARGE B-cell type)
- 44. **Mantle**: MANTLE cell lymphoma (a B-cell NHL)(originates from cells in the MANTLE zones of lymphoid follicles)(nodular follicular pattern on histology)
- 45. Connecting "11" and "14" candles: MANTLE cell lymphoma occurs as a result of a translocation between chromosomes 11 and 14 t(11;14)
- 46. **Cell cycle clock**: in MANTLE cell lymphoma, the t(11;14) translocation results in overexpression of Cyclin D1 (promotes transition from G1 to S phase → uncontrolled cell division)
- 47. **Skinny, sweaty, and flaming bandana**: NHL can present with "B" symptoms (weight loss, fever, night sweats)

Myeloid Disorders 2.4 - Plasma Cell Neoplasms



- "Archery Competition" on BONE sign: in PLASMA CELL NEOPLASMS, a mature B cell gains a mutation → then differentiates into plasma cell clones → plasma cells release their antibodies from the bone marrow
- 2. **Identical archers**: in PLASMA CELL NEOPLASMS, the plasma cells are MONOCLONAL
- 3. **Identical arrows**: in PLASMA CELL NEOPLASMS, monoclonal plasma cells secrete a MONOCLONAL IMMUNOGLOBULIN (termed "M protein")
- 4. "Sir Milo": MULTIPLE MYELOMA (MM) (most common plasma cell neoplasm
- 5. White beard: MM is most common in the elderly (mean age 70)
- 6. **Skeletal clothing**: in MM, clonal plasma cells infiltrate multiple sites throughout the skeletal system
- 7. "M" shaped helmet: in MM, clonal plasma cells synthesize large amounts of monoclonal immunoglobulin ("M" protein)
- 8. Forked arrow : in MM, M proteins are most commonly $\lg G$ (but can be any $\lg type$)
- 9. **Spike on M helmet**: on serum protein electrophoresis (SPEP), the M protein creates a single narrow peak in gamma-globulin region (termed "M spike")
- 10. **LIGHT-colored CHAINmail**: in MM, plasma cells may produce free LIGHT CHAINS (in addition to "M" protein, a complete immunoglobulin with heavy and light chains)
- 11. Urine puddle: free light chains are excreted in urine (due to small size)
- 12. "Sir Jones" on the BENCH : "Bence Jones proteins" (term for free light chains in urine)
- 13. **Holes in spine quiver**: MM presents with lytic bone lesions (most commonly in vertebral column)
- 14. Holes in helmet: MM presents with lytic bone lesions throughout skeleton (skull, ribs, pelvis, femur, clavicle, scapula [in addition to vertebral column]) (causes pathologic fractures)
- 15. **Grimacing from torn spine quiver**: MM can present with back pain induced by movement or position changes (due to vertebral compression fractures)
- 16. **Hunched over from spine quiver**: MM can present with height reduction (due to vertebral compression fractures)
- 17. Holes in skull & crossed arrows flag: in MM, X-ray demonstrates "punched-out" lesions in affected bone
- Destructive osteoCLASS mates: in MM, lytic bone lesions are due to increased OsteoClast activity (bone resorption) AND decreased osteoblast activity
- 19. "RANK L RANK": lytic bone lesions may be due to expression of RANKL (receptor activator of nuclear factor kappa-B ligand) on osteoblasts → binds RANK on osteoclasts → activates osteoclasts to increase bone resorption

- 20. **Raising milk bottles**: MM can present with HYPERcalcemia (increased bone resorption & decreased bone formation → increased serum calcium levels)
- 21. **Ghost white** : MM can present with normochromic normocytic anemia (bone marrow replacement by tumor cells \rightarrow suppression of normal RBC production)
- 22. **Bacterial lanterns**: MM may present with recurrent infections (Staph aureus, Strep pneumoniae, E coli) (neoplastic cells inhibit function of normal plasma cells → decreased production of normal functional antibodies)
- 23. Armoured lady with LIGHT chainmail & beta pleated skirt: MM can cause AL amyloidosis (amyloid composed of fragments of immunoglobulin LIGHT chains)
- 24. Cracked kidney shield: MM can present with renal insufficiency (due to a combination of Bence Jones proteins, direct toxicity, amyloidosis & hypercalcemia)
- 25. Bench near cracked kidney shield : in MM, renal insufficiency is partially due to Bence Jones proteins (bind uromodulin in urine \rightarrow precipitate as dense casts \rightarrow obstruct renal tubules)
- 26. Fan Cone near cracked kidney shield: in MM, renal insufficiency is partially due to toxic effect of light chains on proximal tube → generalized reabsorptive defect ("Fanconi syndrome") → excessive excretion of amino acids, glucose, phosphate, bicarb (→ type 2 renal tubular acidosis)
- 27. Chainmail kidney bag: in MM, renal insufficiency is partially due to amyloidosis (deposits as extracellular fibrils in tissue → tissue injury)
- 28. **Milk spilling on kidney shield** : in MM, renal insufficiency is partially due to excess serum calcium (deposits in kidney \rightarrow injury) ("metastatic calcification")
- 29. "10" flag pole & round target (under bone banner): in MM, bone marrow biopsy demonstrates increased numbers of plasma cells (> 10% of bone marrow cells)
- 30. Blue target with purple center: on histology, neoplastic plasma cells appear similar to normal plasma cells
- 31. Outer blue ring; plasma cells display a basophilic cytoplasm due to abundant rough ER (for protein [immunoglobulin] production):
- 32. Pale spot : in plasma cells, cytoplasm contains a pale spot (due to non-staining of golgi apparatus)
- 33. Clock-faced purple circle: in plasma cells, the eccentric nucleus has a "wagon wheel" or "clock-faced" distribution of chromatin
- 34. Stack of red targets: in MM and LPL/WM, the peripheral blood smear may display rouleaux formation (stacked RBCs due to elevated serum protein levels)
- 35. "Sold Out": MM is sensitive to treatment with proteaSOME INHIBitors (borteZOMIB) (because plasma cells synthesize large amount of protein)
- 36. Chopping up proteins in PROTEIN zone: PROTeasomes (filled with proteases) function to degrade cellular proteins



- 37. **Insignificant arrow blown away by wind GUST**: Monoclonal Gammopathy of Undetermined Significance (MGUS)
- 38. "GUS" hoping to become "Sir Milo": minority of MGUS progress to MM, amyloid, WM
- 39. Small "M" spike without armour or assistants: MGUS is characterized by a small M protein spike WITHOUT symptoms of multiple myeloma
- 40. **Knocked down "10" flag and target**: in MGUS, bone marrow biopsy displays < 10% monoclonal plasma cells
- 41. White beard: MGUS is relatively common in the elderly
- 42. **Solitary arrow in the arm**: SOLITARY PLASMACYTOMA (plasma cell neoplasm that affects a single location, most commonly a bone lesion) (NO systemic symptoms)
- 43. **Grimacing**: SOLITARY PLASMACYTOMA affecting a bone can present with pain or pathologic fractures at the site
- 44. **Hoping to become "Sir Milo**: SOLITARY PLASMACYTOMAS can progress to MM
- 45. "Sir Walden": Waldenstrom Macroglobulinemia (WM) (hyperviscosity syndrome caused by LymphoPlasmacytic Lymphoma)
- 46. **Chess board with antibody archers**: LymphoPlasmacytic LYMPHOMA (LPL) is a neoplasm of clonal mature B lymphocytes → some differentiate into monoclonal plasma cells
- 47. IgM ninja star : in LPL, monoclonal plasma cells secrete IgM \rightarrow forms IgM pentamers
- 48. "M" helmet with spike: LPL displays an M-spike in the gammaglobulin region on SPEP (representing clonal IgM)
- 49. "10" flag with target : in LPL, bone marrow biopsy displays > 10% neoplastic cells
- 50. Small purple targets accompanying plasma cell target: in LymphoPlasmacytic Lymphoma (LPL), neoplastic cells in the bone marrow are a combination of Plasma cells AND other Lymphoid cells (small lymphocytes, plasmacytoid lymphocytes)
- 51. **Ninja stars in thick red mud**: LPL causes a hyperviscosity syndrome ("Waldenstrom's Macroglobulinemia") (WM) (due to massive volume of circulating IgM pentamers)

- 52. **Hit in the helmet**: WM can present with headaches, dizziness, tinnitus, deafness, confusion (due to sluggish blood flow in the CNS)
- 53. **Oversized glasses with blue cracks**: WM can present with visual impairment (sluggish blood flow in retinal veins → dilated tortuous veins → abnormal vision)
- 54. **Gloves & stockings**: WM can present with peripheral neuropathy (symmetrical sensory) (due to sluggish blood flow in peripheral nervous system)
- 55. **Easy bleeding**: WM can present with bleeding diathesis (excessive IgM interferes with clotting factors and platelet function)
- 56. **Ninja stars in "Cold Drinks" box**: WM can present with cryoglobulinemia (due to precipitation of IgM pentamers at low temperatures)
- 57. **Blue fingers**: cryoglobulinemia can manifest as Raynaud phenomenon, urticaria, purpura, or even tissue necrosis
- 58. White round bells: LymphoPlasmacytic LYMPHOMA (LPL) can present with lymphadenopathy (due to infiltration of lymph nodes by neoplastic cells)
- 59. Liver & spleen spots: LymphoPlasmacytic LYMPHOMA (LPL) can present with hepatosplenomegaly (due to infiltration of liver and spleen by neoplastic cells)
- 60. **Ghost white** : LPL can present with anemia (bone marrow replacement by tumor cells \rightarrow suppression of normal RBC production)
- 61. **Straining ninja stars**: in LPL/WM, severe hyperviscosity is treated with plasmapheresis

Immunology 1.1 - Autoimmune Disease: Overview & Systemic Lupus Erythematosus (SLE)





- 1. Familiar hallway: autoimmunity (immune system inappropriately reacting to self-antigens)
- 2. **High School of Los Angeles** : some Human Leukocyte Antigen (HLA) alleles confer genetic susceptibility for autoimmune diseases (includes HLA-B27 [a MHC Class I allele] and HLA-DR & HLA-DQ [MHC Class II alleles])
- 3. Locker TRIGGERing mockery: environmental triggers for autoimmune diseases
- 4. Bacterial & viral ribbons: infections (viral or bacterial) can trigger autoimmunity (foreign antigens incite immune system to react against self-antigens)
- 5. **Mimicking mirror**: infections may trigger autoimmunity via MOLECULAR MIMICRY (viruses or bacteria present antigens that are similar to self-antigens → immune system targets self [in addition to virus/bacteria]) (M protein in Strep pyogenes → rheumatic heart disease)
- 6. Mortar & pestle decal: drugs can trigger autoimmunity (procainamide, hydralazine, isoniazid [drug-induced lupus])
- 7. **Toxin decal** : toxins can trigger autoimmunity (cigarette smoke → rheumatoid arthritis)
- 8. Exposing secret diary : autoimmunity can be triggered by release of normally SEQUESTERED antigens → immune system views "new" antigen as foreign → immune response (example: intracellular antigens exposed as result of cell damage)
- 9. **Teen wolf girl**: systemic lupus erythematosus (SLE) (multisystem autoimmune disease) (most commonly presents in young women [age 20s-30s])
- 10. "DR Q": HLA-DR and HLA-DQ alleles (encode for MHC Class II molecules) increase susceptibility to SLE
- 11. **Female bathroom**: estrogens may contribute to the development of SLE (by stimulating the cell-mediated immunity) (explains increased incidence in females)
- 12. AUTO-shop : formation of auto-antibodies is a crucial for pathogenesis of SLE
- 13. Antibody clamps on car nucleus : SLE presents with anti-nuclear antibodies (ANAs) (directed against antigens that are normally sequestered in cell nucleus)
- 14. Double-stranded cables on antibody clamps : SLE frequently presents with anti-double stranded DNA antibodies (specific for SLE) (an ANA)
- 15. "Always be SPECIFIC": anti-double stranded DNA antibodies are highly specific for SLE
- 16. **Mr Smith**: SLE may present with anti-Smith antibodies (Smith antigen: the core protein of small nuclear ribonucleoproteins [snRNPs]) (specific for SLE) (an ANA)
- 17. **Terminals A & B (on duRO LAst battery)**: SLE may present with anti-Ro (SSA) and anti-La (SSB) antibodies (more common in Sjogren's Syndrome) (an ANA)
- 18. "U" car logo & "Runs on Natural Petroleum" sticker: SLE may present with anti-U1 RNP antibodies (more common in mixed connective tissue disease) (an ANA)
- 19. "Don't do DRUGS, you'll be HISTORY": DRUG-induced lupus may present with anti-HISTONE antibodies (highly specific) (an ANA)
- 20. **Tired & wrapped joints**: DRUG-induced lupus presents with arthralgias [painful joints] and fatigue (no involvement of skin, kidneys, or CNS)
- 21. Eating red circular meat with antibody fork: SLE may present with antibodies against RBCs
- 22. Pale kid: SLE may present with autoimmune hemolytic anemia (due to antibodies directed against RBCs)
- 23. Plate with antibody fork: SLE may present with antibodies against platelets
- 24. **Broken plates**: SLE may present with immune thrombocytopenia (due to antibodies directed against platelets)
- 25. Antibody fork on "white squire" school logo : SLE may present with antibodies against lymphocytes

- 26. **Torn squire mascot**: SLE may present with decreased lymphocyte count (specifically decreased regulatory T cells) (due to antibodies against lymphocytes)
- 27. Antibody earrings paired with phospholipid necklace: SLE may present with antiphospholipid antibodies → causes antiphospholipid syndrome
 28. Overflowing thrombotic trash: antiphospholipid syndrome is characterized by
- 28. Overflowing thrombotic trash: antiphospholipid syndrome is characterized by a hypercoagulable state (increased risk of blood clots & pregnancy loss)
- 29. **Sports COMPLEX with inflammatory torch & 3 pillars** : in SLE, ANAs bind their antigens to form antigen-antibody immune COMPLEXES \rightarrow deposit throughout body \rightarrow inflammatory response in the affected tissue (type 3 hypersensitivity reaction)
- 30. "High five": in SLE, antigen-antibody immune complexes activate the classical COMPLEMENT pathway \rightarrow activate secretion of inflammatory molecules \rightarrow tissue injury
- 31. **Torn "3-4 PM" poster**: active SLE presents with decreased C3 and C4 (due to consumption of complement proteins)
- 32. Falling thin kid with flame bandana : SLE can present with constitutional symptoms (fatigue, fever, weight loss)
- 33. **Glomerular bendy straw**: SLE can present with glomerulonephritis (deposition of immune complexes within glomeruli \rightarrow inflammation \rightarrow proliferation of endothelial, mesangial, or epithelial cells) (presents with oliguria, hematuria, hypertension, RBC casts in urine)
- 34. **Butterfly-patterned rouge**: SLE can present with a malar rash ("butterfly" distribution) (acute cutaneous lupus erythematosus) (may present months or years before systemic symptoms)
- 35. **Discoid red sunglasses**: SLE may present with a discoid rash (round scaly erythematous plaques and follicular plugging) (chronic cutaneous lupus erythematosus)
- 36. Standing near UV window : SLE-associated skin rashes are exacerbated by UV light ("photosensitivity")
- 37. **Drippy skull pattern between upper and lower shirt**: in SLE, affected skin displays liquefactive degeneration and edema at the dermoepidermal junction
- 38. **Glowing green skulls**: in SLE, immunofluorescence of affected skin reveals deposition of Ig and complement at dermoepidermal junction
- 39. **Migrating athlete with wrapped hands and knees**: SLE commonly presents with arthritis (migratory symmetrical polyarthritis) affecting hands and knees
- 40. **Small torn fibers**: SLE can present with vasculitis (due to immune complex deposition in blood vessels) most commonly involving cutaneous small vessels
- 41. Palpating small purple bite marks : cutaneous small vessel vasculitis presents with palpable purpura
- 42. Fingerless gloves with black nail polish : SLE can present with Raynaud phenomenon (cold temperature \rightarrow vasospasm \rightarrow pale cyanotic digits)
- 43. Inflamed pack lining : SLE can present with pericarditis (due to immune complex deposition within the pericardium)
- 44. Shark-tooth necklace : pericarditis presents with sharp precordial chest pain (worsened with inspiration, relieved by leaning forward)
- 45. **Lunch Sack**: SLE can present with Libman-Sacks endocarditis (sterile verrucous endocarditis) (aggregations of immune complexes, leukocytes, and
- 46. **2-cusped plastic bag**: Libman-Sacks endocarditis most commonly affects the MITRAL valve
- 47. **Brain beanie (near torn vascular stockings)**: SLE can present with CNS involvement (cognitive impairment, strokes, seizures) (small vessel vasculopathy → microscopic ischemia and infarcts)
- 48. Streak of black polish : SLE can present with stroke

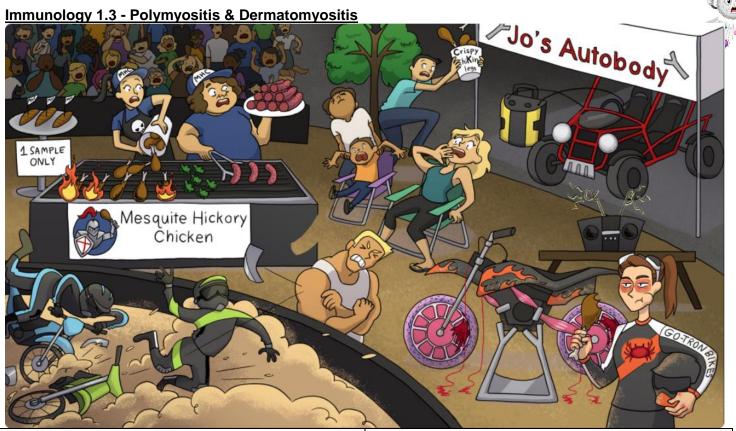
Immunology 1.2 - Sjogren's Syndrome, Systemic Sclerosis & Mixed Connective Tissue Disease





- 1. Female Sjogren warrior: as with most autoimmune conditions, Sjogren syndrome is much more common in women (as high as 9:1 in Sjogren syndrome)
- Dry desert eyes: keratoconjunctivitis sicca (severely dry eyes characteristic of Sjögren syndrome
- 3. **Dry cloth covering mouth**: xerostomia (severe dry mouth characteristic of Sjogren's syndrome)
- 4. Cracks in glandular stones : damage to exocrine glands (from infection or genetic mutation) may lead to the release of self antigens→ development of selfreactive lymphocytes
- 5. **Boy in white holding torch**: the pathogenesis of Sjogren's syndrome involves T and B cells reactive to self antigens→ autoimmune response → severe inflammation
- 6. "XeRo" in sign: anti-SSA (Ro) antibodies are a serologic marker of Sjogren's syndrome (though they do NOT cause direct damage)
- 7. "Lands" in sign: anti-SSB (La) antibodies are a serologic marker of Sjogren's syndrome (though they do NOT cause direct damage)
- 8. "A" tent marker: anti-SSA (Ro) antibodies are a serologic marker of Sjogren's syndrome (though they do NOT cause direct damage)
- 9. "B" tent marker : anti-SSB (La) antibodies are a serologic marker of Sjogren's syndrome (though they do NOT cause direct damage)
- 10. Pumice stones near eyes and mouth on camel : Sjogren syndrome most commonly affects the lacrimal and salivary exocrine glands
- 11. Blue bugs swarming camel's eyes and mouth : there is an intense lymphocytic infiltrate in the lacrimal and salivary glands in Sjogren's syndrome
- 12. **Helper boy in white leading came!** : the lymphocytic infiltrate in Sjogren's syndrome is primarily CD4+ helper T cells and plasma cells
- 13. Purple patches on camel's neck : extensive lymphocytic infiltration→ formation of germinal centers WITHIN salivary glands (germinal centers are the sites of B cell maturation and are usually ONLY found in lymphoid tissue)
- 14. Cracked pumice stones near camel's eye and mouth: lymphocytic infiltration causes effacement and destruction of normal glandular tissue
- 15. **Dry, eroding painted eyes** : lacrimal gland destruction \rightarrow lack of tears \rightarrow drying, inflammation, and erosion of the cornea (keratoconjunctivitis sicca)
- 16. Dry, peeling paint on mask : salivary gland destruction \rightarrow lack of saliva \rightarrow severe drying oral mucosa (xerostomia)
- 17. **Breaks in mouth paint**: severe drying of the oral mucosa (xerostomia) can lead to ulceration, fissuring, and dental caries
- 18. **Dry, tattered loincloth**: destruction of Bartholin's glands (exocrine glands of the vagina) can lead to vaginal dryness, dyspareunia (painful intercourse), and increased infection (both upper and lower urinary tract)
- 19. **Holding arthritis lantern**: Sjogren syndrome often has overlapping features of other autoimmune conditions (especially rheumatoid arthritis)
- 20. Chessboard in camp : chronic lymphocytic infiltration and inflammation \rightarrow increased risk of non-Hodgkin B cell lymphoma
- 21. Scaley dragon: diffuse systemic sclerosis
- 22. Cressandei the lesser : limited systemic sclerosis (CREST syndrome)
- 23. **Dermarys the QUEEN**: like other autoimmune diseases, systemic sclerosis predominantly affects adult women
- 24. **Damaged endothelial tiles**: pathogenesis of systemic sclerosis may involve endothelial damage (possibly from viral infection or toxins) → release of self antigens → self-reactive lymphocytes (molecular mimicry)
- 25. Desert boy in white offering coins : endothelial damage \rightarrow cytokine release inflammation and CD4+ helper T cell infiltration

- 26. **Cartilaginous sharks on shield**: inflammation and cytokine release (including IL-13 and $TGF-\beta$) \rightarrow fibroblast activation \rightarrow excessive collagen production and fibrosis)
- 27. **Fibrotic dead vegetation** : excessive collagen production \rightarrow fibrosis and destruction of native tissue
- 28. **Scaly, narrowed tail** : repeated episodes of inflammation and endothelial damage \rightarrow intimal fibrosis \rightarrow narrowing of small vessel lumen
- 29. Charred skeleton : intimal fibrosis \rightarrow narrowing of small vessel lumen \rightarrow distal ischemic injury
- 30. Antibody hair clips and unwinding hair: anti-DNA topoisomerase I (ScI-70) autoantibodies are the serologic marker of diffuse systemic sclerosis (may be present in some cases of limited sclerosis) (they do NOT cause direct damage)
- 31. **Dragonfly antibody staff**: anti-centromere antibodies most specific for limited sclerosis (some patients may have anti-topoisomerase I (ScI-70) antibodies) (neither cause direct damage)
- 32. **Diffusely scaly dragon**: skin changes (progressive fibrosis → thickening and loss of elasticity) are the most common feature of systemic sclerosis
- 33. **Dragon claws**: (sclerodactyly) progressive fibrosis of the hands and fingers \rightarrow thin, claw like fingers with skin that often ulcerates and limits motion in joints
- 34. Red vascular jewel surrounded by sapphires : systemic sclerosis often shows perivascular CD4+ lymphocytic infiltrate on skin biopsy
- 35. Totally pink patterned outfit : systemic sclerosis causes collagen deposition and fibrosis throughout the entire dermis— thick, tightened skin
- 36. **Bones buried in sand**: Nodular subcutaneous calcification (calcinosis cutis) may occur in systemic sclerosis (especially limited sclerosis)
- 37. Fingerless gloves with blue nail polish : Raynaud phenomenon (cold or stress \rightarrow arterial vasoconstriction \rightarrow pallor or cyanosis in the digits)
- 38. **Choked by rope**: the most common GI finding in systemic sclerosis is esophageal hypomotility→ dysphagia (though any part of the GI tract may be involved)
- 39. **Fibrotic lung tree**: systemic sclerosis may cause interstitial lung disease or intimal fibrosis and narrowing of pulmonary vessels → pulmonary hypertension
- 40. **Dragon blowing steam** : renal arterial intimal fibrosis and vessel narrowing → decreased renal perfusion → activation of RAAS → hypertension (sometimes severe)
- 41. Black heart collar : recurrent vasospasm of small coronary vessels \to myocardial ischemia and patchy fibrosis ("cardiac Raynaud")
- 42. Corked heart jug : pulmonary hypertension \to right heart strain \to right ventricular hypertrophy and failure (cor pulmonale)
- 43. Cressandei's CREST : CREST syndrome (limited sclerosis) Calcinosis cutis, Raynaud, Esophageal hypomotility, Sclerodactyly, Telangiectasias
- 44. **Blood red highlights in Cressandei's hair**: telangiectasias are cutaneous lesions formed by dilated blood vessels, especially on the face, upper trunk, and hands (more common to CREST syndrome than diffuse systemic sclerosis)
- 45. **Defecting sorceress**: like most autoimmune conditions, mixed connective tissue disease most commonly affects adult women
- 46. **Mixed fabric outfit**: mixed connective tissue disease share features of many autoimmune disorders, including SLE, systemic sclerosis, rheumatoid arthritis, and many others
- 47. **Crescent moon antibody staff**: anti-U1 ribonucleoprotein antibodies are seen the serologic marker of mixed connective tissue disease (though they do NOT cause direct damage)



- 1. Abundant grilled meat : POLYMYOSITIS (inflammatory myopathy)
- 2. **Female grillmasters** : polymyositis & dermatomyositis are more common in women
- 3. Abundant "MHC" flags on surface of striped meat: pathogenesis of polymyositis begins with INCREASED expression of MHC I on the sarcolemma (cell membrane of striated muscle cells)
- 4. **Cytotoxic T cell knight wielding fried chicken**: in polymyositis, CD8+ cytotoxic T cells initiate myofiber destruction (increased expression of MHC I \rightarrow presentation of autoantigens to cytotoxic T cells \rightarrow activation)
- 5. **Fiery meat**: in polymyositis and dermatomyositis, muscle biopsy demonstrates inflammation
- 6. **Skull apron**: in polymyositis and dermatomyositis, muscle biopsy demonstrates myocyte necrosis
- 7. Regenerating chicken legs: in polymyositis and dermatomyositis, muscle biopsy demonstrates regeneration of muscle fibers
- 8. **Fibrotic vegetables**: in polymyositis and dermatomyositis, muscle biopsy demonstrates fibrosis
- 9. Muscle cell sausages each surrounded by blue peppered skin: in polymyositis, muscle biopsy displays an ENDOmysial inflammatory infiltrate (inflammatory cells surrounding each muscle fiber WITHIN the fascicle)
- 10. **Unable to pick-up child**: polymyositis and dermatomyositis present with symmetric proximal muscle weakness (affecting upper & lower extremities) (includes weakness of deltoids → difficulty raising arms above head)
- 11. **Unable to rise from chair**: in polymyositis and dermatomyositis, weakness of hip flexors (as part of proximal muscle weakness) \rightarrow difficulty rising from chair)
- 12. **Hit by painful debris**: polymyositis and dermatomyositis can present with myalgias
- 13. **Fibrotic lung tree**: polymyositis and dermatomyositis can present with interstitial lung disease
- 14. **Lifting bucket of Crispy chiKin**: polymyositis and dermatomyositis present with elevated Creatine Kinase (marker of muscle cell death)
- 15. **Antibody wrenches on "JO's AUTObody"**: polymyositis and dermatomyositis can present with anti-JO-1 antibodies (also known as anti-tRNA or antisynthetase antibodies)

- 16. **DIRT on muscle bike**: DERMATOMYOSITIS (inflammatory myopathy with significant cutaneous involvement)
- 17. **Broken VASCULAR brake lines on muscle bike**: in dermatomyositis, injury affects MUSCLE fibers AND the CAPILLARIES that feed them
- 18. **Increased radio interference**: pathogenesis of dermatomyositis involves upregulation of type 1 interferon → capillary and myofiber injury
- 19. **Speckled blue tire encircling muscle fascicle wheel**: in dermatomyositis, muscle biopsy displays a PERImysial inflammatory infiltrate (inflammatory cells surrounding the entire muscle fascicle)
- 20. **Focal injury on edge of fascicle wheel**: in dermatomyositis, muscle biopsy displays PERIfascicular muscle cell injury grouped in one portion of fascicle (blood vessel dysfunction → localized microinfarcts)
- 21. **Roughed-up knuckles**: dermatomyositis can present with violaceous papules over joints and bony prominences (particularly dorsal metacarpophalangeal and interphalangeal joints of hands) (Gottron's papules)
- 22. "Go-Tron Bikes" : dermatomyositis can present with Gottron's papules
- 23. **Purple goggle rash**: dermatomyositis can present with a violaceous rash on upper eyelids "heliotrope rash"
- 24. Cancer crab logo on chest: dermatomyositis is associated with an increased risk of adenocarcinoma (particularly of lungs and ovaries)

Immunology 1.4 - Amyloidosis



- 1. **Armoured lady**: amyloidosis (diseases caused by aggregations of misfolded proteins [amyloid] that deposit in tissues)
- 2. β-pleated skirt: AMYLOID fibers are ALWAYS composed of proteins misfolded into BETA-pleated sheets
- 3. Cumbersome EXTERIOR chainmail impairing normal activity : amyloid accumulates in the EXTRACELLULAR space \rightarrow mass effect that impairs normal tissue function
- 4. Red blood: amyloidosis is identified by staining tissues with Congo Red dye
- 5. **Green bi-re-FRAGRANCE**: in tissues stained with Congo Red, the presence of amyloid fibrils produces apple-green birefringence under polarized light
- 6. Small light-colored chains: AL amyloid (fragments of immunoglobulin LIGHT chains)
- 7. Antibody archer with M spike helmet and spine quiver: MULTIPLE MYELOMA can present with AL amyloidosis (due to overproduction of immunoglobulin light chains)
- 8. **Light chains on archer** : in MULTIPLE MYELOMA, proliferation of neoplastic monoclonal plasma cells → overproduction of immunoglobulin (including LIGHT chains, which can form AL amyloid)
- 9. **Multiple archers shooting antibody arrows**: AL amyloidosis is caused by proliferation of monoclonal plasma cells (neoplastic or not) \rightarrow overproduction of LIGHT chains \rightarrow AL amyloid
- 10. **Head-to-toe in chainmail** : AL amyloidosis is SYSTEMIC (amyloid deposits in tissues throughout body)
- 11. LARGE Kidney chainmail bag : AL amyloidosis commonly affects the KIDNEY \rightarrow abnormally large kidney (amyloid initially deposits in glomeruli) (presents with nephrotic syndrome
- 12. **Chainmail restricting heart design**: AL amyloidosis may affect the HEART → presents with RESTRICTIVE cardiomyopathy (amyloid deposits extracellularly between myocardial fibers)
- 13. Large tongue : AL amyloidosis may affect the TONGUE (amyloid deposits macroglossia)
- 14. **Chainmail gloves**: AL amyloidosis may affect the wrist (amyloid deposits in carpal ligaments → compression of median nerve → carpal tunnel syndrome)
- 15. **Shoulder chainmail**: AL amyloidosis may affect the joints (amyloid deposits in synovial tissues \rightarrow arthropathy in shoulders, knees, wrists, metacarpophalangeal, and proximal interphalangeal joints)
- 16. **Neuronal tassels**: AL amyloidosis can present with neurologic involvement (mixed sensory motor peripheral neuropathy → numbness, paresthesia, pain) (autonomic neuropathy → bowel and bladder dysfunction, orthostatic hypotension)
- 17. Large liver and spleen spots : AL amyloidosis can affect the liver and spleen \rightarrow organ enlargement
- 18. **Bloody sword**: AL amyloidosis can affect blood vessels (more fragile → bleeding even from minor trauma)
- 19. **REACTING to armoured lady**: REACTIVE SYSTEMIC amyloidosis (occurs secondary to chronic inflammatory condition)

- 20. **Chronically inflamed bone lanterns**: REACTIVE SYSTEMIC amyloidosis is most commonly secondary to RHEUMATOID ARTHRITIS (or another chronic inflammatory condition)
- 21. **Two "A" spears**: REACTIVE SYSTEMIC amyloidosis is caused by deposition of AA amyloid (composed of fragments of serum amylase A [SSA] [an acute phase reactant])
- 22. **Kidney chainmail bag**: REACTIVE SYSTEMIC amyloidosis most commonly affects the kidney (--> nephrotic syndrome) (although can affect any tissue)
- 23. Cleaning blood from whole-body chainmail: HEMODIALYSIS-related amyloidosis (a SYSTEMIC amyloidosis)
- 24. **Two \beta daggers**: HEMODIALYSIS-related amyloidosis is caused by amyloid derived from BETA-2 MICROGLOBULIN (components of MHC I molecules) (not efficiently filtered by dialysis \rightarrow accumulation)
- 25. Chainmail gloves and shoulder pads : HEMODIALYSIS-related amyloidosis most commonly affects osteoarticular structures (including shoulders and wrists)
- 26. "Cart Pool Tunnel" : HEMODIALYSIS-related amyloidosis commonly presents with carpal tunnel syndrome (amyloid deposition in carpal ligaments of the wrist \rightarrow median nerve compression \rightarrow carpal tunnel syndrome)
- 27. Elderly man with chainmail headgear : SENILE CEREBRAL amyloidosis (localized amyloidosis)
- 28. Confused : SENILE CEREBRAL amyloidosis is associated with Alzheimer's disease)
- 29. "A β " crown design : SENILE CEREBRAL amyloidosis is caused by A β amyloid \to deposits extra-cellularly in central nervous system
- 30. "APP" crown design : A β amyloid is derived from Amyloid Precursor Protein (APP)
- 31. A β amyloid on thin VASCULAR crown : CEREBRAL AMYLOID ANGIOPATHY (CAA) (caused by A β amyloid deposition in blood vessels of the brain \rightarrow vessel weakening \rightarrow increased risk of spontaneous cerebral hemorrhage)
- 32. **Chainmail kid with flame bandana**: Familial Mediterranean Fever (FMF) (HERITABLE amyloidosis)
- ${\bf 33.} \ \textbf{Receding behind mom}: \textbf{Familial Mediterrane} \textbf{an Fever is autosomal recessive}$
- 34. **Persistently lit PYRE** : Familial Mediterranean Fever is caused by a mutation in the PYRIN gene \rightarrow overproduction of cytokines \rightarrow persistent inflammation
- 35. Family near TWO "A" spears & REACTIVE soldiers: Familial Mediterranean Fever leads to deposition of AA amyloid and presents as REACTIVE SYSTEMIC amyloidosis
- 36. "THY Transporter" : genetically mutated forms of transTHYRETIN (prealbumin) \rightarrow deposition of ATTR amyloid \rightarrow HERITABLE amyloidoses
- 37. **Multiple gold neuronal chain links**: Familial Amyloidotic Polyneuropathies (mutated transthyretin \rightarrow ATTR deposits in peripheral and autonomic nerves \rightarrow motor, sensory, and/or autonomic neuropathies)
- 38. **Gold chainmail heart** : Familial Amyloid Cardiomyopathy (mutated transthyretin \rightarrow ATTR deposits in myocardium \rightarrow cardiomyopathy)
- 39. **Grey chainmail heart** : SYSTEMIC SENILE AMYLOIDOSIS (normal [wild-type] transthyretin aggregates into ATTR amyloid as a result of aging \rightarrow ATTR deposits in myocardium \rightarrow cardiomyopathy

Immunology 2.1 - B-Cell Disorders & DiGeorge Syndrome



- 1. **Crutches at PRIMARY school play**: PRIMARY (inherited) immunodeficiency
- 2. Viral & bacterial hats: primary immunodeficiency increases the risk of infections (bacteria, viral, fungal, and/or protozoal depending on affected immune system component)
- 3. AUTO-reactive antibody hair clips: primary immunodeficiency increases the risk of AUTOimmune disorders (due to defective immune regulatory mechanisms)
- 4. **Crab gaming console**: primary immunodeficiency increases the risk of malignancy (due to decreased clearance of oncogenic viruses, defective surveillance for malignant cells)
- 5. Chess board with B cell archers and T cell knights: in primary immunodeficiency, non-Hodgkin lymphoma is the most common malignancy
- 6. Young child: primary immunodeficiency usually presents with recurrent infections between 6 months 2 years of age
- 7. "Y" antibody pattern passed from mother's dress to baby's blanket : in the 3rd trimester, maternal IgG passess through the placenta to the fetus \rightarrow provides baby with passive immunity for 4-6 months
- 8. "A" antibody pattern on mother's chest : after birth, maternal IgA passes through breast milk to baby \rightarrow provides baby passive immunity during the breastfeeding period
- 9. **"6" hair curl**: primary immunodeficiency typically presents only AFTER 6 months of age because of passive immunity from IgG (lasts 4-6 months) and IgA (lasts only during breastfeeding period)
- 10. **No "LIVE"**: in primary immunodeficiency, LIVE vaccines are contraindicated (even if pathogen is attenuated)

- 11. Great "Bruton" flag: BRUTON DISEASE (X-linked agammaglobulinemia [XLA]) (B cell primary immunodeficiency)
- 12. Moustache falling off antibody bullet-firing soldier : in XLA, PRE-B cells fail to differentiate into MATURE B cells
- 13. **Torn "B"ruton**: in XLA, B cells are nearly ABSENT in peripheral blood (due to failure of differentiation into mature B cells)
- 14. **Dropped antibody bullets** : in XLA, levels of ALL Ig classes are nearly ABSENT (agammaglobulinemia) (no mature B cells \rightarrow no plasma cells \rightarrow no antibody production)
- 15. Receding behind pole with "X" flag : XLA is X-linked recessive
- 16. "TYRE": XLA is caused by a mutation in the Bruton TYROSINE kinase (BTK) gene (encodes a tyrosine kinase that signals pre-B cells to mature)
- 17. Encapsulated food beneath bacterial lanterns: XLA increases susceptibility to ENCAPSULATED BACTERIA (Strep pyogenes, H. influenzae) because normally cleared by antibody-mediated opsonization and phagocytosis (→ recurrent otitis media, sinusitis, pneumonia)
- 18. **GI dolly beneath viral lantern**: XLA increases susceptibility to VIRAL infections (particularly ENTEROvirus)
- 19. **Protozoal hat in gravy puddle**: XLA increases susceptibility to Giardia lamblia (intestinal PROTOZOA usually neutralized by secreted $lgA) \rightarrow persistent diarrhea$
- 20. **"X-GERM" hand sanitizer**: in XLA, lymphoid tissue lacks GERMINAL centers (normally the site of B cell aggregation)
- 21. **Undersized white crumpets**: in XLA, LYMPHOID tissues are abnormally SMALL (due to absence of germinal centers)
- 22. **Infiltrating Canadian soldier**: skin injection of Candida antigens can be used to confirm isolated B cell defect (such as XLA)
- 23. "T" bayonet lighting Canadian soldier on fire: in an isolated B cell defect (such as XLA), skin injection of Candida antigens results in WARM induration (because functioning T cells are responsible for immune response against Candida and other fungi)
- 24. **Submit "CV/ID" here**: COMMON VARIABLE IMMUNODEFICIENCY (CVID) (heterogenous group of B cell primary immunodeficiency disorders)
- 25. A few dropped antibody bullets: CVID is characterized by LOW (but not absent) Ig levels (HYPOgammaglobulinemia)
- 26. Intact mustache: in CVID, a NORMAL number of MATURE B cells are present in peripheral blood
- 27. "Marksmen wanted": in CVID, PLASMA cells are DEFICIENT (due to failure of B cells to differentiate into plasma cells)
- 28. "Adult Men and Women": CVID typically presents in early adulthood (delayed because antibodies are not completely absent)

Immunology 2.1 - B-Cell Disorders & DiGeorge Syndrome



- 29. Large burning "IgA" bridge: SELECTIVE IgA DEFICIENCY (most common primary immunodeficiency) (B cells fail to differentiate into IgA secreting plasma cells)
- 30. **Smiling soldier**: IgA DEFICIENCY is frequently asymptomatic
- 31. **Dirty lung spots**: IgA DEFICIENCY increases susceptibility to sinopulmonary infections (because IgA is the major immunoglobulin in mucosal secretions)
- 32. **Protozoal hat in gravy puddle**: IgA DEFICIENCY increases susceptibility to Giardia lamblia (intestinal PROTOZOA) (because IgA is the major immunoglobulin in mucosal secretions)
- 33. "Draining blood" flag: IgA DEFICIENCY can present with anaphylaxis to BLOOD transfusions (because donated blood normally contains small amount of IgA)
- 34. **Antibody bridge posts**: IgA DEFICIENCY can lead to formation of anti-IgA antibodies (because immune system considers IgA a foreign protein)
- 35. Beehive with histamine bees : in IgA DEFICIENCY, exposure to IgA in transfused blood \rightarrow degranulation of MAST cells containing anti-IgA IgE antibodies \rightarrow ANAPHYLAXIS
- 36. **DiGeorge Washington** : DiGeorge SYNDROME (congenital defect in thymus development \to T cell primary immunodeficiency disorder)
- 37. **Dumping boxes of THYME**: DiGeorge syndrome presents with HYPOPLASIA or APLASIA of the THYMUS gland
- 38. **Dropping "T" bayonet and losing moustache**: in DiGeorge syndrome, T cells are unable to MATURE (because of thymic aplasia) (T cells are produced in the bone marrow, but mature in the thymus)
- 39. **Scratched-out "T3A"**: DiGeorge syndrome presents with ABSENT MATURE (CD3+) T cells (CD3 is a marker of ALL mature T cells)
- 40. **Viral lanterns**: DiGeorge syndrome increases susceptibility to VIRAL infections (requires T cell-mediated immunity)
- 41. **Fungal mushrooms**: DiGeorge syndrome increases susceptibility to FUNGAL infections (requires T cell-mediated immunity)
- 42. **Bacterial lanterns within tent**: DiGeorge syndrome increases susceptibility to INTRAcellular BACTERIAL infections (requires T cell-mediated immunity)
- 43. **Neural crest shield**: DiGeorge syndrome is caused by defective migration of neural crest cells → abnormal development of 3rd and 4th pharyngeal pouches
- 44. **Fallen "TA3" & "TE4" pouches**: DiGeorge syndrome is characterized by abnormal development of 3rd & 4th pharyngeal pouches

- 45. **Tipped-over PthD**: DiGeorge syndrome presents with LOW PARATHYROID hormone (abnormal development of 3rd & 4th pharyngeal pouches → parathyroid gland HYPOplasia)
- 46. **Spilled milk**: DiGeorge syndrome presents with HYPOcalcemia (due to low parathyroid hormone)
- 47. **Abnormal face mask**: DiGeorge syndrome presents with abnormal facies (due to maldevelopment of 3rd & 4th pharyngeal pouches)
- 48. Cleft hat : DiGeorge syndrome presents with cleft palate (due to maldevelopment of 3rd & 4th pharyngeal pouches)
- 49. **Blue heart**: DiGeorge syndrome presents with cyanotic congenital heart disease (maldevelopment of 3rd & 4th pharyngeal pouches → abnormal aortic arch) (truncus arteriosus, tetralogy of Fallot)
- 50. "22" "11" coat pattern: DiGeorge syndrome is caused by deletion of part of chromosome 22 (22q11 microdeletion)
- 51. "22" "11" VELVET coat with intact THYME: 22q11 microdeletion can cause VELOcardiofacial syndrome (similar to DiGeorge except THYMUS is NORMAL)
- 52. Face mask, cleft hat & blue heart: VELOcardiofacial syndrome presents with abnormal facies, cleft palate, and cyanotic congenital heart disease
- 53. **Jolly roger with torn right-sided triangular sail**: DiGeorge syndrome presents with an ABSENT thymic shadow on X-ray (normally seen in infants as a TRIANGULAR SAIL to the right of mediastinum)
- 54. **Torn "PARcel TAX"**: in DiGeorge syndrome, lymph nodes demonstrate a poorly developed PARACORTEX (site where mature T cells normally aggregate)

Immunology 2.2 - Combined B & T-Cell Disorders





- 1. **PRIMARY school kid with crutches**: PRIMARY (inherited) immunodeficiency
- 2. Fallen comic books (with B-cell archer & T-cell knight): combined B-cell & T-cell deficiency
- 3. **Wasps**: Wiskott-Aldrich syndrome (combined B and T cell primary immunodeficiency \rightarrow viral, fungal, encapsulated bacterial infections)
- 4. Boy receding behind "X" door : Wiskott-Aldrich syndrome is X-linked recessive
- 5. Wasp stings: Wiskott-Aldrich syndrome presents with eczema
- 6. **Broken plates**: Wiskott-Aldrich syndrome presents with thrombocytopenia
- 7. **Disorganized cytoskeleton nest** : mutations in Wiskott-Aldrich Syndrome Protein lead to ABNORMAL actin CYTOSKELETON reorganization → impaired T cell function (including abnormal regulation of B cells) → B cell dysfunction
- 8. Bacterial lanterns with encapsulated pizza: Wiskott-Aldrich syndrome increases susceptibility to ENCAPSULATED BACTERIA (Strep pneumo, H. influenzae, N. meningitidis) because clearance requires antibodies (→ recurrent otitis media, sinusitis, pneumonia, meningitis)
- 9. **Viral lanterns**: Wiskott-Aldrich syndrome increases susceptibility to VIRAL infections (CMV, Varicella zoster)
- 10. **Fungal mushrooms**: Wiskott-Aldrich syndrome increases susceptibility to FUNGAL infections (candida, PJP)
- 11. $\bf Small~`GaMes"~sign~:$ in Wiskott-Aldrich syndrome, serum IgG & IgM are LOW to NORMAL
- 12. **Elevated "EAt" sign** : in Wiskott-Aldrich syndrome, serum IgA & IgE are ELEVATED
- 13. "Hyper coMics" : Hyper-IgM syndrome (combined B and T cell primary immunodeficiency \rightarrow viral, fungal, encapsulated bacterial infections)
- 14. Raised "M" cards with fallen "A" "G" "E": in Hyper-IgM syndrome, B cells produce IgM but NOT IgA, IgG, or IgE (because B cells are unable to switch isotypes)
- 15. Cracked glass over "40 thieves" poster: Hyper-IgM syndrome is caused by a mutation in the gene encoding CD40 LIGAND on helper T cells \rightarrow B cells unable to bind helper T cells (requires CD40) \rightarrow no B cell isotype switching
- 16. **Boy receding behind "X" door**: Hyper-IgM syndrome is X-linked recessive
- 17. "X-GERM" hand sanitizer: in Hyper-IgM syndrome, lymphoid tissue lacks GERMINAL centers (normally the site of mature B cell proliferation, differentiation, and isotype switching)
- 18. **SKID marks**: Severe Combined ImmunoDeficiency (SCID) (constellation of syndromes resulting in severe combined B and T cell primary immunodeficiency → viral, fungal, encapsulated bacterial infections)

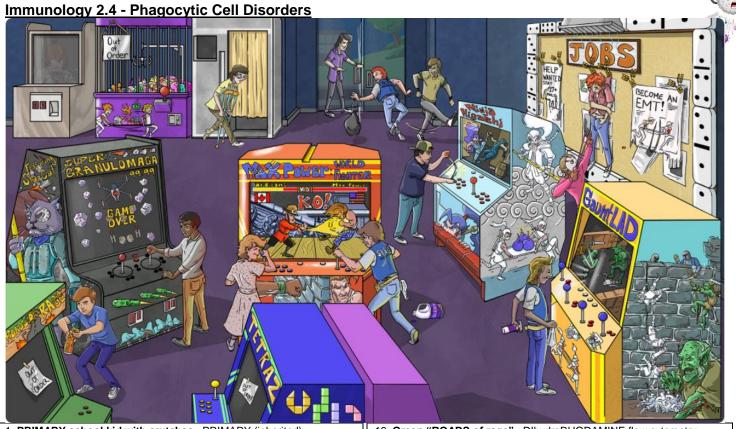
- 19. **Boy receding behind "X" bike**: X-LINKED recessive mutation in cytokine receptors (including IL-7) (most common cause of SKID)
- 20. Holding back"11-7" soda : mutations in interleukin-7 (IL-7) lead to DECREASED numbers of MATURE B and T cells \to SCID
- 21. Receding behind car: AUTOSOMAL recessive mutation in ADA (causes SCID
- 22. Beat-up bADAss plate & purine logo : mutations in Adenosine Deaminase (ADA) lead to abnormal PURINE metabolism \rightarrow DECREASED B and T cells \rightarrow SCID
- 23. **Spilling antibody keys**: in SCID, ALL types of immunoglobulins are LOW (SPEP displays minimal peak in gamma globulin region) because mature B cells are absent
- 24. **Scratched-up T-3IRD logo**: in SCID, T cells are LOW to absent (flow cytometry displays minimal CD3 peak [CD3 is marker on ALL mature T cells])
- 25. **Fallen "THYME" cookbook** : SCID presents with THYMIC hypoplasia (due to absent mature T cells)
- 26. **Jolly roger with torn right-sided triangular sail**: SCID presents with an ABSENT thymic shadow on X-ray (normally seen in infants as a TRIANGULAR SAIL to the right of mediastinum)
- 27. **"X-GERM"** hand sanitizer: in SCID, lymphoid tissue lacks GERMINAL centers (normally the site of B cell aggregation)
- 28. **Tipped over ATM** : Ataxia-Telangiectasia (combined B and T cell primary immunodeficiency \rightarrow primarily encapsulated bacteria susceptibility \rightarrow recurrent sinopulmonary infections)
- 29. **Receding behind ATM**: Ataxia-Telangiectasia is caused by AUTOSOMAL recessive mutation in Ataxia Telangiectasia Mutated (ATM) gene
- 30. Broken surveillance camera & damaged double helical wires : mutations in ATM lead to impaired surveillance of DNA damage \rightarrow B and T cell deficiency
- 31. **Unbalanced body**: Ataxia-Telangiectasia presents with ataxia (due to cerebellar atrophy)
- 32. **Disorganized red wires**: Ataxia-Telangiectasia presents with telangiectasias in the skin (abnormal capillary dilatations)
- 33. Scratched-out "T": in Ataxia-Telangiectasia, T cells are LOW
- 34. **Falling "A"**: in Ataxia-Telangiectasia, serum IgA is LOW (because B cells are unable to isotype switch to IgA)
- 35. "Alf's Fresh Produce" in high window : in Ataxia Telangiectasia, serum alpha-fetoprotein (AFP) is ELEVATED

Immunology 2.3 - Complement System Disorders



- 1. **PRIMARY school kid with crutches**: PRIMARY (inherited) immunodeficiency
- 2. **Swollen lip**: Hereditary Angioedema (primary immunodeficiency due to complement defect)
- 3. **Broken "no C1hatting" sign**: Hereditary Angioedema is caused by C1 INHIBITOR DEFICIENCY → UN-inhibited C1 → unchecked cleavage (activation) of downstream complement proteins (C2, C4)
- 4. **OVERSIZED complimentary "high five"**: Hereditary Angioedema leads to an OVERACTIVE complement system (due to decreased inhibition of C1)
- 5. Domino pattern: Hereditary Angioedema is autosomal dominant
- 6. Broken "NO Kalling" sign : in Hereditary Angioedema, deficient C1 inhibitor \rightarrow REDUCED inactivation of KALLIKREIN
- 7. **Uncontrolled BRAIDS**: in Hereditary Angioedema, BRADYKININ production is UNCONTROLLED (deficient C1 inhibitor → increased kallikrein activation → increased conversion of kininogen to bradykinin)
- 8. **Seats "3a, 4a, 5a"**: in Hereditary Angioedema, complement proteins C3a, C4a, C5a are ELEVATED (due to overly active complement cascade)
- 9. **Dilated red sleeves** : in Hereditary Angioedema, abundant VASOACTIVE substances (bradykinin, C3a, C4a, C5a) \rightarrow vasodilation and increased vascular permeability
- 10. **Highly swollen lip**: Hereditary Angioedema presents with widespread angioedema (painless, non-pitting swelling of face, neck, lips, tongue)
- 11. Clutching belly: Hereditary Angioedema can present with abdominal pain (due to angioedema in GI tract)
- 12. **Green face**: Hereditary Angioedema can present with nausea/vomiting (due to angioedema in GI tract)
- 13. **Spilled brown soda**: Hereditary Angioedema can present with diarrhea (due to angioedema in GI tract)
- 14. Falling "C 4II-Star" sneaker : in Hereditary Angioedema, C4 levels are LOW (deficient C1 inhibitor → uninhibited C1 → consumption of C4)
- 15. **Silencing ACE cellphone**: in Hereditary Angioedema, ACE inhibitors are contraindicated (ACE normally degrades bradykinin) (inhibited ACE → increased BRADYKININ → increased angioedema)

- 16. **Cracked "1, 2, 3, 4" countdown timer**: C1, C2, C3, C4 deficiencies (primary immunodeficiencies)
- 17. **Highlighted "2"**: C2 deficiency is the most common complement deficiency
- 18. Encapsulated bacterial light: C1, C2, C3, C4 deficiencies increase susceptibility to ENCAPSULATED BACTERIA (Strep pneumo, H. influenzae, N. meningitidis) because early complement system is required to opsonize foreign pathogens for phagocytosis
- 19. "Wolf Teen" : C1, C2, C4 deficiencies increase the risk of systemic lupus erythematosus $\,$
- 20. Torn "5-9": C5-9 (MAC) deficiencies (primary immunodeficiencies)
- 21. "MAC Private Eye": Membrane Attack Complex (MAC, composed of C5-C9) inserts into pathogen's cell membrane \rightarrow opens pores that disrupt osmotic balance \rightarrow pathogen lysis
- 22. "Noir series": C5-C9 deficiencies increase susceptibility to Neisseria species (particularly meningitidis) (because MAC is crucial for neutralizing Neisseria)
- 23. **Peaceful Night Hotel** : Paroxysmal Nocturnal Hemoglobinuria (PNH) (deficiency in complement inhibitor proteins \rightarrow INTRAvascular hemolysis)
- 24. Broken anchor for "PIG"-A sign : in PNH, mutations in the PIG-A gene \rightarrow deficient GPI anchor protein \rightarrow decreased complement inhibitors (CD55 and CD59) on RBC surface
- 25. Fallen "Route 59" and Route "55" signs : in PNH, DECREASED complement inhibitors (CD55 and CD59) on RBC cell surface \rightarrow overactive complement system (including MAC) \rightarrow RBC lysis
- 26. **Broken hemoglobin hubcaps on vascular road**: PNH presents with intravascular hemolysis (due to RBC lysis by complement system)



- 1. **PRIMARY school kid with crutches**: PRIMARY (inherited) immunodeficiency
- 2. **EMT first responders on "Out of Order" machine** : NEUTROPHIL dysfunction \rightarrow impaired PHAGOCYTOSIS \rightarrow defect in innate immunity
- 3. MacroCAGE on "Out of Order" machine : MACROPHAGE dysfunction \rightarrow impaired PHAGOCYTOSIS \rightarrow defect in innate immunity
- 4. "Super Granulomaga": Chronic Granulomatous Disease (CGD) (primary phagocytic disorder) (impaired degradation of ingested microbes within the phagolysosome)
- 5. "X"-shaped ship: CGD is most commonly X-linked recessive
- 6. **High-energy electrolyte drink**: NADPH is an electron carrier (normally oxidized to produce NADP+ and reactive oxygen species)
- 7. **Unable to remove "H" cap** : CGD is caused by a deficiency in NADPH oxidase \to NADPH is not converted into NADP+ \to no production of oxygen free radicals
- 8. **2** "O" joysticks & red electron button: normally, an electron released by oxidation of NADPH is donated to oxygen \rightarrow forms a SUPEROXIDE ion (O2-) [as part of the RESPIRATORY BURST in the phagolysosome]
- "H-OO-H" ship: normally, superoxide is converted into HYDROGEN PEROXIDE [as part of the RESPIRATORY BURST in the phagolysosome]
- 10. **Cracked "SUPER"**: in CGD, no NADPH oxidase → no release of oxygen free radicals → no SUPERoxide → minimal hydrogen peroxide → minimal RESPIRATORY BURST
- 11. Featuring "Star Cat": CATALASE breaks down hydrogen peroxide into oxygen and water
- 12. **Bacterial ships & fungal mushrooms**: many bacteria and ALL fungal pathogens are CATALASE positive → very effective at breaking down hydrogen peroxide
- 13. **Golden staff**: CGD predisposes to infections with CATALASE positive organisms (most commonly Staph Aureus) (the small amount of hydrogen peroxide in a CGD phagolysosome is broken-down by catalase → NO respiratory burst)
- 14. **Rusty chest plate**: CGD commonly presents with pneumonia (due to infections with CATALASE positive organisms)
- 15. **MULTI-spotted granular asteroids**: CGD can present with GRANULOMAS in any organ (may contain MULTINUCLEATED giant cells) (malfunctioning phagocytes release inflammatory mediators \rightarrow T cells and macrophages organize into granulomas)

- 16. **Green "ROADS of rage"**: DlhydroRHODAMINE flow cytometry (presence of SUPEROXIDE converts dihydrorhodamine to rhodamine [a fluorescent green compound])
- 17. "Roads" Out of Order: in CGD, the DIhydroRHODAMINE test produces MINIMAL GREEN fluorescence (absence of SUPEROXIDE → no conversion of dihydrorhodamine to rhodamine)
- 18. **Blue "TETRAZ"** : nitroBLUE TETRAzolium (NBT) test (exposure of NBT to SUPEROXIDE \rightarrow production of BLUE dye)
- 19. "TETRAZ" Out of Order: in CGD, the NBT test produces MINIMAL BLUE dye (due to absence of SUPEROXIDE
- 20. **Broken "Max Power"**: MYELOPEROXIDASE (MPO) Deficiency (primary phagocytic disorder) (impaired degradation of ingested microbes within the phagolysosome)
- 21. "Max Power" starting to die: in the phagolysosome, MyeloPeroxidase converts hydrogen peroxide to hypochlorous acid (BLEACH, kills everything!)
- 22. **Dropped empty BLEACH** : in MPO deficiency, the phagolysosome does not produce hypochlorous acid (BLEACH) \rightarrow impaired degradation of ingested microbes
- 23. Fighter receding behind wall : MPO deficiency is AUTOSOMAL recessive
- 24. Canadian "Albicans" : MPO deficiency predisposes to recurrent Candida infections
- 25. **Intact BURST**: in MPO deficiency, the respiratory burst (production of superoxide and hydrogen peroxide) is INTACT



- 26. **GauntLAD**: Leukocyte Adhesion Deficiency (LAD) (primary phagocytic disorder) (impaired extravasation of leukocytes from blood into tissues)
- 27. Falling white leukocyte army: in LAD, leukocytes are unable to adhere to blood vessel walls \to no migration into tissues
- 28. Receding behind castle wall: LAD is AUTOSOMAL recessive
- 29. Scratched out "18+" : LAD results from deficiency of CD18 \rightarrow interferes with formation of integrins \rightarrow impaired adhesion of leukocytes to endothelial wall
- 30. **Skin boils & ulcers**: LAD presents with recurrent bacterial infections (particularly in skin and mucosal surfaces) (skin abscesses, otitis media)
- 31. **Toothless gums**: LAD presents with loss of adult teeth by adolescence (due to periodontitis)
- 32. **Sparkling clean**: in LAD, infections lack purulence (leukocytes unable to migrate to site of infection \rightarrow no leukocyte death \rightarrow no pus)
- 33. **Unable to sever rope**: LAD presents with delayed separation of the umbilical cord (no migration of leukocytes into the umbilical stump \rightarrow no weakening of connecting tissues \rightarrow cord remains in place)
- 34. **Pile of white soldiers**: LAD presents with leukocytosis even in the absence of infection (because leukocytes are trapped in circulation) (increases during infection)
- 35. **First responders**: in LAD, leukocytosis is primarily composed of neutrophils

- 36. "Jobs" board stapled with IgE pins: Hyper-IgE (Job) syndrome (primary phagocytic disorder) (impaired neutrophil chemotaxis)
- 37. Domino border: Hyper-IgE syndrome is autosomal dominant
- 38. **Torn "STAT" sign**: Hyper-IgE syndrome is caused by a mutation in the STAT3 gene (part of the JAK-STAT signal transduction pathway)
- 39. 17+ only: in Hyper-IgE syndrome, Th17 Helper T cells are deficient (due to mutations in STAT3)
- 40. **Torn "EMT" recruitment poster** : in Hyper-IgE syndrome, neutrophil chemotaxis is impaired (STAT3 mutations → deficient Th17 helper T cells → decreased secretion of cytokines for neutrophil chemotaxis and proliferation)
- 41. **Abnormally COLD air vent**: Hyper-IgE syndrome presents with cold skin abscesses & absence of fever during systemic infection (lack of neutrophil chemotaxis → absent inflammatory response to infection)
- 42. **Crusty acne and dandruff**: Hyper-IgE syndrome presents with recurrent skin infections (begins in newborns with pruritic papulopustular crusted rash on face and scalp)
- 43. **Hockey mask**: Hyper-IgE syndrome can present with coarse facies (broad nasal bridge, deep set eyes)
- 44. **Slingshot loaded with pink granules**: in Hyper-IgE syndrome, eosinophil count may be elevated]
- 45. "Ninja Higashi": Chediak Higashi syndrome (primary phagocytic disorder) (defective fusion of phagosomes and lysosomes)
- 46. Receding behind wall: Chediak Higashi syndrome is AUTOSOMAL recessive
- 47. **Trash disrupted en route to dumpster**: in Chediak Higashi syndrome, lysosomal TRAFFICKING is disrupted → lysosome is unable to fuse with phagosome → phagocytosed microbe never reaches lysosome → microbe is not destroyed
- 48. **First responders with large blue bombs**: in Chediak Higashi syndrome, neutrophils contain giant azurophilic granules
- 49. **Golden staff**: Chediak Higashi syndrome presents with recurrent infections with Staph aureus and other pyogenic bacteria
- 50. Completely white ninja: Chediak Higashi syndrome presents with oculocutaneous albinism
- 51. Zapped by broken wires: Chediak Higashi syndrome presents with peripheral neuropathy (motor and sensory)

Musculoskeletal & Derm 1.1 - Osteoporosis & Paget Disease of Bone





- 1. Young Australopithecus lifting stone: bone mass increases in childhood and peaks around the second to third decade
- 2. Line over cow falling after peak: following peak bone mass development, bone resorption starts to exceed bone formation (typically after 40)
- 3. **Old osteobuilder taken over by osteoCLAST class**: senile osteoporosis is due to osteoCLAST activity overtaking osteoBLAST activity→ bone resorption and demineralization (typically in the eighth decade)
- 4. **Exercising skeleton**: decreased weight-bearing activity contributes to senile osteoporosis
- 5. PAUSED female on video screen : loss of estrogen after menopause → increased osteoCLAST activity→ bone resorption greater than formation
- 6. CytoCOINS spilling from donation box : loss of estrogen after menopause→ release of inflammatory cytokines (IL-1, IL-6, TNF-alpha) → increased bone turnover
- 7. Kid stealing RANK-L crank drill from osteobuilder: inflammatory cytokines increase Receptor Activator of Nuclear Factor k Ligand (RANK-L) expression on osteoblasts, which activates RANK receptors on osteoclasts→increased bone resorption
- 8. RANK-L drill falling out of OPG protective case : inflammatory cytokines decrease production of osteoprotegerin (which inhibits osteoclasts) by osteoblasts → uninhibited osteoCLAST activity → increased bone resorption
- 9. Flattening spongy trabecular ottoman : osteoporotic trabecular bone becomes thinner with fewer connections between trabeculae→ "hollow" appearance
- 10. **Drilling into long bone column**: osteoporotic cortical bone shows cortical thinning and widened Haversian canals (blood vessel "canals" in lamellated bone)
- 11. **ROWDY kid holding back in pain**: compression fractures of the vertebral column are the most common fragility fractures in osteoporosis
- 12. **Humped spine**: excessive kyphosis caused by anterior vertebral body compression leads to a stooped posture called "Dowager's Hump"
- 13. **Fractured hand holding fork**: falling on an outstretched hand can cause a distal radial fracture (Colles' fracture) that has a "dinner fork" appearance on x-ray (dorsal displacement of distal radial fragment)
- 14. ROWDY kid breaking off femur : fracture of the surgical neck of the femur can occur following mild trauma (fall from standing) in severe osteoporosis
- 15. **Bone inDEX book**: Dual-energy X-ray Absorptiometry (DEXA) scanning is used to diagnose osteoporosis and osteomalacia
- 16. **Fractured bone under bone index table**: fragility fractures, even with a T score over -2.5, are diagnostic of osteoporosis
- 17. **Smoker**: smoking inhibits osteoblast activity, increasing risk of osteopenia and osteoporosis
- 18. **Drinking from flask**: heavy alcohol use is a risk factor for osteopenia and osteoporosis

- 19. CLASSmate with RANK-L drill under steroid moon : steroids increase production of RANK-L (stimulates osteoclasts) and decrease production of osteoprotegerin (inhibits osteoclasts) → increased osteoclastic bone resorption
- 20. Broken estrogen necklace under moon : steroids decrease estrogen production \rightarrow increased bone resorption
- 21. **Sitting on cushion under moon**: excess adrenal cortisol production in Cushing syndrome can cause osteopenia and osteoporosis
- 22. **Mortar and pestle**: drugs such as heparin, H2 blockers, proton pump inhibitors, and anticonvulsants such as phenytoin and carbamazepine are associated with osteopenia and osteoporosis
- 23. **PThD curator**: excess PTH in hyperparathyroidism stimulates osteoCLASTS→ increased bone resorption→ osteopenia and osteoporosis
- 24. **Big red bowtie**: excess thyroid hormone due to hyperthyroidism or excess levothyroxine intake stimulates osteoclasts→ increased bone resorption→ osteopenia and osteoporosis
- 25. Osteo-CLASSmates grabbing bones from Paget box : the first phase of Paget's disease is the osteoLYTIC phase→ extensive osteoclastic activity causing bone resorption and formation of resorption pits
- 26. **Disorganized Paget skeleton**: the second phase of Paget's disease is mixed→ osteoblast and osteoclast activity cause rapid bone turnover resulting in disorganized bone formation
- 27. Osteobuilders fixing Paget skeleton : the third phase of Paget's disease is osteosclerotic→ osteoblasts form bulky, fragile bone
- 28. Pink mosaic rug under Paget skeleton : disorganized "mosaic" woven and lamellar bone formation with haphazardly arranged cement lines is seen in Paget's disease
- 29. Paget's problematic parts: the skull, femur, and pelvic bones are most commonly affected in Paget's disease
- 30. **Tiny hat big skull**: skull enlargement is a classic finding in Paget's disease (patients often complain of hats not fitting anymore)
- 31. **Trying to listen to Paget skull**: cochlear involvement in Paget's disease can lead to hearing loss
- 32. **Broken chalk** : abnormally remodeled bone in Paget's disease is weak and prone to "chalk-stick" fractures→ bone pain
- 33. **High output headlamp with floppy heart balloon**: metabolic demand in early Paget's disease causes extensive hypervascularity→ arteriovenous shunts that can lead to high-output heart failure
- 34. Cancer crab speared by Paget skeleton: long standing Paget's disease is a risk factor for osteosarcoma (though risk is small, typically <1%)
- 35. **Box of chALK**: increased osteoblast activity results in elevated alkaline phosphatase levels in Paget's disease
- 36. **Skull and crossbones by shaggy hair kid**: x-ray findings in Paget's disease include "shaggy" dark areas of radiolucency (early disease) and abnormally thick bone (late disease)

Musculoskeletal & Derm 1.2 - Osteomalacia & Rickets



- Soft pink rock: the first step of bone formation is osteoblastic production of osteoid, a proteinaceous matrix composed of most type I collagen
- 2. **Fish bones**: maturation of bone involves deposition of calcium phosphate into osteoid, which is converted to organized hydroxyapatite
- 3. **Map of Malaysia**: osteoMALACIA is due to decreased MINERALIZATION of osteoid→ accumulation of unmineralized collagen matrix with normal bone mass (opposed to low bone MASS in osteoporosis)
- 4. **Broken shark jaw bone**: rickets (seen in children) is due to defective CARTILAGE mineralization in growth plates (as opposed to osteoid mineralization in osteomalacia)
- 5. **2 sharks on broken plate**: rickets is due to decreased mineralization of TYPE II HYALINE CARTILAGE of epiphyseal growth plates (found in long bones)
- 6. Bones and phosphate fossils by fallen "D": vitamin D deficiency leads to calcium and phosphate deficiency→ impaired mineralization of bone→ osteomalacia (adults) and rickets (children)
- 7. Vitamin D feast : vitamin D is found naturally in foods like fatty fish and in fortified products like milk, cheese, orange juice, and cereals
- 8. Always in the moonlight: UV light exposure converts 7-dehydrocholesterol to calcitriol in skin; lack of sunlight can lead to vitamin D deficiency
- 9. **Pigmented skin**: naturally pigmented skin contains more melanin→ decreased UV light absorption and calcitriol production→ higher risk of vitamin D deficiency
- 10. **Kidney shaped egg fossils**: chronic kidney disease results in deficiency of 1-alpha hydroxylase→ decreased activation and deficiency of vitamin D (1-alpha hydroxylase converts 25-hydroxyvitamin D to active 1,25-dihydroxyvitamin D)
- 11. **Chunky water leaking**: malabsorption syndromes (gastric bypass, IBD, chronic pancreatitis) can lead to vitamin D deficiency
- 12. **Fallen ADEK sign** : malabsorption of fat (chronic pancreatitis, IBD, surgery) can lead to deficiency of fat-soluble vitamins A, D, E, and K

- 13. **PthD picking up bones**: hypocalcemia stimulates release of PTH (secondary hyperparathyroidism) → bone resorption to raise serum calcium→ demineralization of bone
- 14. PthD leaving phosphate fossils down : PTH increases calcium REABSORPTION and phosphate EXCRETION in the kidney— increased calcium and decreased phosphate
- 15. **chALK board**: continued osteoblast activity leads to elevated alkaline phosphatase in both osteomalacia and rickets
- 16. **Security guard knee pain**: osteomalacia commonly causes bone pain worsened by weight bearing activity
- 17. **Fractured bone flashlight**: deficient mineralization of bone in osteomalacia and rickets makes them weak and prone to fracture (especially in vertebrae and femoral neck)
- 18. **Shadowy wrinkles in pirate flag**: pseudofractures ("Looser zones") are radiolucent (dark) lines resembling bone fractures commonly seen on x-ray in osteomalacia and rickets
- 19. **Outward bowing table legs**: toddlers with rickets often develop outward bowing of the legs (varus bowing)
- 20. **Knee high pottery table**: older children with rickets often develop inward, "knock-knee" bowing of the legs (valgus bowing)
- 21. **Shark plush hat**: infants with rickets can develop craniotabes (abnormal soft areas of skull bone) as well as frontal or parietal bossing (protruding skull bone)
- 22. **Non-fused turtle shell**: infants with rickets often have delayed closure of fontanelles
- 23. Wide, frayed open bone basket: widening of the epiphyseal growth plate and fraying of the metaphysis are commonly seen on x-ray in rickets
- 24. **Open basket on shorter table**: decreased cartilage mineralization of open epiphyseal growth plates→ short stature in children with rickets
- 25. Rachitic rosary necklace: enlargement of the costochondral junction forms nodules at the ends of ribs that can be seen on CXR or palpated on the anterior chest wall ("rachitic rosary")

Musculoskeletal & Derm 1.3 - Benign Bone Tumors



- 1. Happy fraternity member: BENIGN bone tumors (usually asymptomatic) (most common in males, late teens or twenties)
- 2. Barrier hedge blocking stray balls: BENIGN bone tumors do NOT invade local structures, destroy the cortex, or invade the growth plate
- 3. **OC surfer dude**: OsteoCHONDROMAS (most common benign bone tumor) (primarily occurs in males age 20's)
- 4. Metaphyseal bulge of long bone tree : OsteoCHONDROMAS most commonly grow out of endochondral tissue at the METAPHYSIS $\,$
- 5. **Tree bud**: OsteoCHONDROMAS present as an EXOSTOSIS (outgrowth of bone) either sessile (no stalk) or pedunculated (with a stalk)
- 6. Ball stuck in stagnant mud: OsteoCHONDROMAS grow slowly
- 7. Shark cap : OsteoCHONDROMA cells produce cartilage \rightarrow tumors are covered with a CARTILAGE CAP
- 8. **Mallet over knee**: OsteoCHONDROMAS are most common in distal femur (near the KNEE)
- 9. Painful break : OsteoCHONDROMAS can become PAINFUL if the stalk BREAKS \rightarrow trauma in surrounding soft tissue (otherwise tumor is painLESS)
- 10. **Small crab**: OsteoCHONDROMAS are associated with a SMALL chance of transformation into MALIGNANT osteosarcoma
- 11. **Middle aged "Mister Oste"**: OSTEOMA (benign bone tumor) (most common in middle aged males)
- 12. Bulge in cortical rocks : OSTEOMAS are composed of CORTICAL bone
- 13. **Hit in the face**: OSTEOMAS commonly grow in bones of HEAD & NECK (particularly within nose and sinuses)
- 14. **Garden Lilipolyps**: OSTEOMAS are associated with GARDNER polyposis syndrome (also associated with colonic polyps, supernumerary teeth, desmoid tumors)
- 15. **Omega Omega**: OSTEOID Osteoma (benign bone tumor) (most common in males age 20's)
- 16. Sitting on outer house layer : OSTEOID Osteomas typically develop in the bone CORTEX
- 17. Sun tan: OSTEOID Osteomas are composed of well-defined TAN tissue

- 18. Jolly roger: X-ray
- 19. White framed sunglasses : on X-ray, OSTEOID Osteomas display a radiolucent area surrounded by a bright sclerotic ring of reactive cortical bone
- 20. **Sunglass case on upper thigh**: OSTEOID Osteomas are most common in the PROXIMAL FEMUR
- 21. **Pain while wearing sleepy cap**: OSTEOID Osteomas can present with NOCTURNAL bone pain (due to production of prostaglandins)
- 22. **Anti-inflammatory fire extinguisher**: in OSTEOID Osteomas, pain is RELIEVED by NSAIDS (because originates from prostaglandin production by tumor)
- 23. Large tan "Osteo Builder" : OsteoBLASTOMA (benign bone tumor) (most common in males in age 20's) (large [>2 cm] TAN tumor)
- 24. **Swinging-back spinal mallet**: OsteoBLASTOMAS are most common within POSTERIOR SPINAL COLUMN
- 25. **Back pain**: OsteoBLASTOMAS can present with focal neurological symptoms or PAIN (large tumors → spinal cord compression) (NOT relieved by NSAIDS)
- 26. **END of the Contest**: ENChondromas (benign bone tumor) (affect men and women equally) (most common in adolescents)
- 27. Shark shirt: EnChondromas produce CARTILAGE
- 28. **Bone stake with medullary line**: EnChondromas develop in the MEDULLARY CAVITY (location of bone marrow)
- 29. Croquet gloves & shoes : EnChondromas are most common in hands and feet $\,$
- 30. Large croquet ball: GIANT CELL tumor of bone (benign bone tumor) (most common in women between ages 20s-40s)
- 31. Ball at EPIPHYSIS of bone mallet: GIANT CELL tumor typically involves EPIPHYSIS of long bones
- 32. Mallet swinging past knee: GIANT CELL tumor most commonly occurs near KNEE (distal femur or proximal tibia)
- 33. **Aggressively bending wicket**: GIANT CELL tumors can be locally aggressive
- 34. **Giant purple spots**: in GIANT CELL tumor, large multinucleated osteoclast GIANT cells are interspersed within neoplastic stromal cells

Musculoskeletal & Derm 1.4 - Malignant Bone Tumors



- 1. **Infiltrating the party via bone banister**: the most common cancer in bone is METASTASIS (most commonly PB-KTL: prostate, breast, kidney, thyroid, lung)
- 2. **Butt hitting bannister**: PROSTATE cancer commonly metastasizes to the bone
- 3. Crab bra: BREAST cancer commonly metastasizes to the bone
- 4. Kidney purse: RENAL cancer commonly metastasizes to the bone
- 5. **Bow tie**: THYROID cancer commonly metastasizes to the bone
- 6. Crab shirt: LUNG cancer commonly metastasizes to the bone
- 7. Bone & Crab party table : OSTEOsarcoma (most common malignant bone tumor)
- 8. **Raising Root Beer**: Hereditary Retinoblastoma increases the risk of OSTEOsarcoma (mutation in RB1 tumor suppressor)
- 9. **So FrauMany Varieties**: Li-Fraumeni syndrome increases the risk of OSTEOsarcoma and many other cancers
- 10. **Broken checkpoint gate**: Li-Fraumeni syndrome is caused by a mutation in the tumor suppressor gene TP53 (normally functions as a cell cycle checkpoint protein)
- 11. **Radiated bone**: RADIATION therapy (particularly in children) increases the risk of OSTEOsarcoma
- 12. **Bony "Discarded" pile**: OSTEOsarcoma in older patients (age >40) is commonly due to sarcomatous transformation of PAGET's Disease of the bone (characterized by DISORGANIZED bone growth)
- 13. **Pools of PINK slime** : on histology, OSTEOsarcomas display PINK-staining osteoid
- 14. Whitish mass with bloody spots: OSTEOsarcomas form a gritty white mass with cystic degeneration and areas of hemorrhage
- 15. Bone split open to display marrow : OSTEOsarcomas often invade the MEDULLARY cavity
- 16. **Crab on the knee**: OSTEOsarcoma is most common near the KNEE (distal femur and proximal tibia)
- 17. Jolly roger: X-ray
- 18. **Sunburst window**: on X-ray, OSTEOsarcoma displays a "SUNBURST" pattern (represents spiculated osteoid with patches of cortical destruction)
- 19. **FISH costume with TRIANGLE head fin**: on X-ray, OSTEOsarcoma & EWING sarcoma display "CODMAN TRIANGLE" (tumor breaks through cortex → raises periosteum away from bone → periosteum calcifies in corner to create a triangle) (can also be caused by OTHER bone tumors)

- 20. Chest littered with crab: OSTEOsarcomas commonly metastasize to lungs (hematogenously) (may have even spread by time of diagnosis)
- 21. "WINGS": EWING sarcoma (malignant bone tumor) (primarily in Caucasians <20 years of age)
- 22. "ECTO": EWING sarcoma originates from neuroECTOdermal tissue
- 23. "33" Jersey (22+11): EWING sarcoma is caused by translocation of the EWSR1 gene (chromosome 22) to the FLI1 gene (chromosome 1) \rightarrow EWS-FLI fusion protein
- 24. Ball popping painfully: EWING sarcoma presents with localized PAIN and SWELLING
- 25. **Ball wedged between mid-arm & mid-leg**: EWING sarcoma is most common in the DIAPHYSIS of long bones in the EXTREMITIES
- 26. Inner red bone plate with escaping wings : EWING sarcoma starts in the MEDULLARY cavity → spreads to invade cortex and periosteum
- 27. **Tan-white pile with black spots & BBQ sauce**: EWING sarcoma appears tan-white with spots of necrosis and hemorrhage
- 28. Blue ventilation dots : on histology, EWING sarcoma displays sheets of small, round blue cells
- 29. **Blue-dotted basketball rim**: on histology, EWING sarcoma displays circular groups of cells with an area of central clearing (Homer-Wright rosettes)
- 30. **Moth-eaten jolly roger**: on X-ray, EWING sarcoma displays a "MOTH-EATEN" appearance (due to small areas of lucency in medullary cavity)
- 31. **Onion ring** : on X-ray, EWING sarcoma displays "ONION PEEL" appearance (tumor growth \rightarrow multiple cycles of periosteal reaction \rightarrow rings of thin layers of bone
- 32. **Crab littering chest**: EWING SARCOMA commonly metastasizes to the lungs (hematogenously)
- 33. **Inflatable shark eating crab**: CHONDROsarcoma (malignant bone tumor) (secretes CARTILAGE)
- 34. **Older guy**: CHONDROsarcomas usually occur OVER age 20 (most commonly >50 years)
- 35. Inner red bone plate : CHONDROsarcoma starts in the MEDULLARY cavity \rightarrow spreads to invade cortex and periosteum
- 36. **Shark drooling pink blobs**: on histology, CHONDROsarcomas display lobules of CARTILAGE invading bone marrow (because tumor secretes cartilage)
- $37. \ \textbf{Crab littering chest}: \ CHONDROs arcomas commonly metastasize to the lung (hematogenously)$

Musculoskeletal & Derm 1.5 - Congenital Bone Disorders: Osteogenesis Imperfecta, Achondroplasia & Osteopetrosis



- 1. Cartilaginous shark sword with "1" handle: Type 1 COLLAGEN (present in scar tissue, bones, tendons, ligaments, skin, sclerae)
- 2. Bomb fuse with 3 strands (2 blue & 1 red): Type 1 collagen forms a triple helix (composed of two alpha-1 chains & one alpha-2-chain)
- 3. **Brittle osteowalker**: OSTEOGENESIS IMPERFECTA (OI, "brittle bone disease") (congenital defect in type 1 collagen)
- 4. Domino armor: OI is most commonly autosomal DOMINANT
- 5. **Brittle cracking bones** : OI presents with FRACTURES with even minimal force (abnormal collagen \rightarrow decreased bone flexibility \rightarrow brittle and weak bones
- 6. Mother with 2 babies: TYPE 2 OI (most severe form)
- 7. **ABNORMAL triple helix fuse** : TYPE 2 OI is caused by mutations that disrupt the formation of the type 1 collagen triple helix \rightarrow ABNORMAL type 1 collagen
- 8. Cracked shark sword : TYPE 2 OI results in many many fractures starting in utero \rightarrow not compatible life
- 9. Osteowalker standing at NORMAL triple helix : In TYPE 2 OI, type 1 collagen is NORMAL, but the AMOUNT is DECREASED \rightarrow less severe disease
- 10. Blue eyes : OI presents with BLUE sclera (decreased collagen \rightarrow more translucent sclera \rightarrow underlying BLUE-gray choroidal veins are visible)
- 11. Covering ears: OI can present with conductive HEARING loss (due to abnormal formation, fracture, or dislocation of ossicles within middle ear)
- 12. **Small teeth** : OI can present with "DENTINOGENESIS imperfecta" (abnormal type 1 collagen \rightarrow abnormal dentin \rightarrow small opalescent teeth that wear out quickly)
- 13. "Chondrion" dwarf: ACHONDROPLASIA (congenital defect of bone formation) (most common form of dwarfism)
- 14. **Shark & bone shield**: ENDOCHONDRAL ossification (mechanism for long bone formation) (chondrocytes produce cartilage mold → osteoblasts replace cartilage with bone)
- 15. **Shield PLATE**: at the epiphyseal GROWTH PLATES, endochondral ossification of long bones continues throughout childhood
- 16. **Cracked sharks**: ACHONDROPLASIA is caused by inherited defect in CHONDROCYTE proliferation → defective endochondral ossification & abnormal epiphyseal growth plate function
- 17. "FriGid FoRtr3ss" & "pull 4 entry": ACHONDROPLASIA is caused by a mutation in Fibroblast Growth Factor Receptor 3 (located on chromosome 4)
- 18. Tire swing: FGFR3 is a Tyrosine Kinase Receptor

- 19. **Pulling excessively**: in ACHONDROPLASIA, Gain of Function mutation in FGFR3 → excessive inhibition of chondrocytes → prevents elongation of bones produced by endochondral ossification (long bones) (bones that grow by intramembranous ossification (skull) are unaffected)
- 20. Domino armor: ACHONDROPLASIA is autosomal DOMINANT
- 21. **Short extremities**: ACHONDROPLASIA presents with short arms & legs, short fingers, bowing of the legs
- 22. Large helmet protruding at forehead : ACHONDROPLASIA presents with macrocephaly (large head) with frontal bossing
- 23. Helmet flattening face & nose] → ACHONDROPLASIA presents with saddle-nose deformity (flattening of nose) and midface hypoplasia :
- 24. **Dysfunctional wall maintenance crew**: OSTEOPETROSIS (congenital bone disorder) (characterized by dysfunctional bone MAINTENANCE)
- 25. Wall-dissolvers abandoning acid pot : in OSTEOPETROSIS, osteoCLASTS are unable to generate an acidic environment \rightarrow unable to dissolve hydroxyapatite \rightarrow unable to resorb bone
- 26. **Builders haphazardly adding marble bone**: in OSTEOPETROSIS, ("marble bone disease") unopposed osteoBLAST activity leads to formation of thick, dense unorganized bone
- 27. **Cracked marble slab**: OSTEOPETROSIS presents with frequent FRACTURES (due to weakness of thick unorganized bone)
- 28. **Pelvis/spine/skull flagpole**: OSTEOPETROSIS most commonly affects PELVIS, SPINE, and SKULL
- 29. Flag of wide bones with bright center : on X-ray, OSTEOPETROSIS displays wide bones with concentric rims ("bone-within-bone") and excess growth at the ends ("Erlenmeyer flask deformity")
- 30. **Nearly empty pan**: OSTEOPETROSIS can present with PANCYTOPENIA (due to bone expansion into marrow)
- 31. Liver & spleen spots: OSTEOPETROSIS can present with HEPATOSPLENOMEGALY (bone expansion into marrow → extramedullary hematopoiesis in liver and spleen)
- 32. **Broken helmet on myelinated rope**: OSTEOPETROSIS can present with cranial nerve compression (optic nerve involvement \rightarrow vision loss) (CN VIII involvement \rightarrow hearing loss) (CN VII involvement \rightarrow facial paralysis)
- 33. Adult protected by domino shield : MILD OSTEOPETROSIS is autosomal DOMINANT and presents in adolescence or adulthood
- 34. Blue baby near pan: INFANTILE Malignant OSTEOPETROSIS is often fatal in infancy (due to severe leukopenia)
- 35. Receding behind wall: INFANTILE Malignant OSTEOPETROSIS is autosomal RECESSIVE

Musculoskeletal & Derm 2.1 - Osteoarthritis

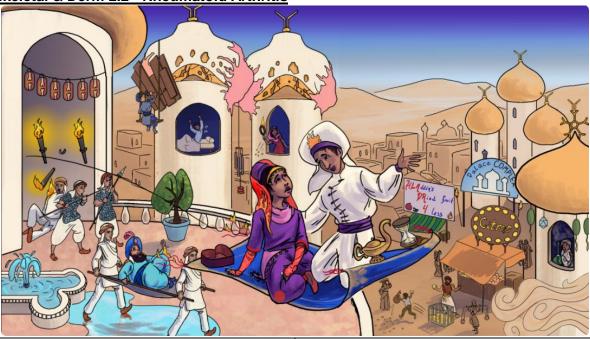


- 1. **Bone scaffold**: Subchondral Bone Plate (area of bone that lies beneath articular cartilage)
- 2. **Scaffold joint**: Articular Cartilage (overlies subchondral bone plate) (provides smooth surface for gliding movements of the joint)
- 3. 2 shark tattoos] \to Articular Cartilage contains a matrix of Type II Collagen (secreted by Chondrocytes within cartilage :
- 4. **Synovial straps**: Synovial Membrane lies within the joint capsule (cells produce Synovial Fluid that fills joint space)
- 5. **HIGHIy carried ACID**: Hyaluronic Acid is present in synovial fluid (lubricates joint)
- 6. Damaged scaffold joint: OSTEOARTHRITIS (OA)
- 7. CytoCOINS released from damaged satchel : OA starts as a result of release of cytokines from chondrocytes → injures articular cartilage
- 8. Proliferation of activated guards : cytokines \rightarrow Proliferation and Activation of chondrocytes
- 9. **Leaking more cytoCOINS**: activated chondrocytes release more cytoKINES
- 10. **METAL swords attacking scaffold joint**: activated chondrocytes → release of Matrix Metalloproteins (MMP) → Break Down extracellular matrix (including type II Collagen)
- 11. **Slow CONTAINED fiery torches**: OA develops as a result of chronic LOCAL inflammation (cycle of cytokine release → chondrocyte activation → release of more cytokine and metalloproteinases → cartilage degradation)
- 12. **Falling shark-tattoo**: as arthritis progresses, MMPs continue to Damage Cartilage matrix and incite chondrocyte death (despite attempts by chondrocytes to produce new cartilage
- 13. **Sclerotic bony snake**: OA can present with Subchondral Sclerosis (inflammation of subchondral bone plate → increased bone turnover → Increased Bone Density and Thickening)
- 14. Ivory helmet protruding from bony club : OA can present with Osteophytes ("Bony Spurs", protrusions of bone and cartilage) (attempts by chondrocytes to repair damaged cartilage → reactive bone)
- 15. **Holes in bone scaffold**: OA can present with Subchondral Cysts (fluid-filled cavitary lesions) (due to cystic degeneration of subchondral bone)
- 16. **Enlarged knees**: OA can present with Enlarged Joints (ongoing inflammation → Thickening of Joint Capsule)

- 17. Fiery PAN with layer of GRease: OA can present with Pannus (layer of GRanulation tissue inside the joint capsule) (due to ongoing inflammation)
- 18. Releasing cytoCOINS : Pannus formation leads to more cytoKINE release \to further degradation of articular cartilage
- 19. Collapsing scaffold joint : OA can present with Joint Space Narrowing
- 20. Old villain: age is a risk factor of OA (usually age > 50)
- 21. Large woman: obesity is a risk factor of OA
- 22. Pain induced by broken broomstick: prior traumatic joint injury is a risk factor for OA
- 23. Broken hip scaffold: OA commonly affects the hip joints
- 24. Swaying spine tree: OA commonly affects lower cervical and lumbar spine
- 25. **Protruding branch stumps** : OA can cause Osteophytes in spine \to impingement of spiral foramina \to Nerve Root Compression
- 26. **Wrapped fingers**: OA commonly affects Finger Joints (proximal [PIP] and distal [DIP] interphalangeal joints)
- 27. **Wrapped thumb**: OA commonly affects Base of Thumb (1st carpometacarpal joint)
- 28. **Unaffected second hand**: OA typically presents with Asymmetric joint involvement
- 29. **Moving stiffly and painfully**: OA presents with Pain and Stiffness in affected joints
- 30. **Sleepy cap**: OA symptoms are typically worse in the Evening (after a day of prolonged use & weight bearing activities)
- 31. **Sleeping peacefully**: OA symptoms Improve with Rest (morning stiffness is rare and improves quickly)
- 32. Creaking scaffold joint: OA can present with Crepitus (crunching or crackling of joint)
- 33. Gnarly staff with 2 bulges: OA can present with swollen nodules on DIP (Heberden's) and PIP (Bouchard's) joints
- 34. **Stuck joints**: advanced OA can present with Decreased Range of Motion (with both active and passive movement)
- 35. **Grounded CARpet**: in OA, C-reactive protein (CRP, acute phase reactant) is NOT elevated (due to lack of systemic inflammation)
- 36. **Grounded ESR wind chime**: in OA, erythrocyte sedimentation rate (ESR, acute phase reactant) is NOT elevated (due to lack of systemic inflammation)

Musculoskeletal & Derm 2.2 - Rheumatoid Arthritis





- 1. **Burning all over** : rheumatoid arthritis (RA) is a disease of SYSTEMIC inflammation
- 2. **Autoantibody adornments** : rheumatoid arthritis is an autoimmune systemic inflammatory disease
- 3. "HLAddin's DRied fruit 4 less": genetic predisposition to RA is evidenced by its strong association with HLA-DR4 alleles (which encode MHC antigen presenting proteins)
- 4. Smoking hookah: smoking is an environmental risk factor for RA
- 5. **Viral and bacterial lanterns**: bacterial or viral infection may expose the immune system to antigens resembling self-antigens→ development of autoimmunity (molecular mimicry)
- 6. **MacroCAGE citrus salesman**: peptidylarginine deaminase (expressed in macrophages, pathogens, and synovial tissue) converts environmental antigen arginine residues to citrulline→ antigen presentation and activation of CD4+ helper T cells
- 7. Citrus thief in white shirt dropping cytoCOINS : after exposure to citrullinated antigens, CD4+ helper T cells infiltrate joints (which contain abundant citrullinated type 2 collagen) \rightarrow cytokine release and inflammation
- 8. Antibody citrus sign holders: anti-citrulline CD4+ helper T cells activate plasma cells— development of anti-cyclic citrullinated peptide antibodies (ACPA) (specific to RA)
- 9. IgM Roof Feature : rheumatoid factor, an IgM antibody that binds the Fc region of IgG, is commonly formed in RA $\,$
- 10. **EMTs servants in puddle**: synovial fluid from joints in RA typically has a neutrophil count >2000 (especially during flares), but well under that of a septic joint (>25,000)
- 11. **Grease fire in pan**: inflammation in RA leads to synovial pannus formation (hyperplasia and granulation tissue formation in joints)
- 12. **Pink gunk engulfing towers** : fibrovascular synovial hyperplasia (pannus) tissue invades surrounding tissue in rheumatoid joints→ loss of cartilage and bone
- 13. Cracks in orange separating layer : active joint inflammation in RA→ production of proteases→ degradation of articular cartilage
- 14. **Holes in white stone** : inflammation in RA activates osteoclasts→ increased bone resorption→ osteopenia and subchondral cyst formation in joint bones
- 15. **Reinforcing synovial tower**: chronic pannus formation and fibrosis in RA can lead to stiffened, near-fused joints (fibrous ankylosis)
- 16. **Small space in tower**: RA causes joint space narrowing, bony erosions, soft tissue swelling, subchondral cysts, and osteopenia
- 17. **Red gloves covering proximal hand**: RA classically causes SYMMETRIC pain and inflammation of the metacarpophalangeal and PROXIMAL interphalangeal joints of the hand

- 18. **Red scarf, gloves, and shoes**: RA typically affects the cervical spine, shoulders, elbows, knees, ankles, and feet (diffuse, symmetric synovial joint involvement)
- 19. **Flame pattern on hats** : RA causes SYSTEMIC inflammation→ fever, malaise, weight loss (vague B symptoms)
- 20. **Painful morning stretch**: patients with RA have joint pain and stiffness worse in the MORNING that typically lasts > 1 hour
- 21. Whistling through the workday: joint pain and stiffness typically IMPROVE with activity in RA (opposed to osteoarthritis, in which pain WORSENS with activity
- 22. **Swan neck lamp**: "swan neck" deformity of the fingers (caused by flexion at the DIP and extension at the PIP joints) is a classic finding in RA
- 23. **Boutonniere buttons**: "Boutonniere" deformity of the fingers (caused by extension of the DIP and flexion of the PIP joints) is a classic finding in RA (REVERSE of "swan neck" deformity)
- 24. Fanning fingers towards ulna: ulnar deviation of the fingers (curvature toward the pinky) is a classic finding in RA
- 25. Wearing hat toward back of head: RA involvement of the cervical spine can lead to cervical instability→ subluxation (displacement of vertebrae) → neck pain, spinal injury, or vertebral artery injury
- 26. **Stiff neck**: fusion of the cervical vertebral facets (ankylosis) can lead to a stiff, immobile neck
- 27. **Fibrotic lung bush**: systemic inflammation in RA can lead to interstitial lung disease
- 28. Velvety heart box : systemic inflammation in RA can lead to pericarditis
- 29. Fiery red tassels : systemic inflammation in RA can lead to vasculitis
- 30. **Stark white shirt**: systemic inflammation in RA can lead to anemia of chronic disease
- 31. **Falling sand granules**: Felty's syndrome is a severe form of RA that causes splenomegaly and decreased production of granulocytes→ neutropenia
- 32. **Nodular rings**: a dermatologic manifestation of RA is formation of rheumatoid nodules (firm, non-tender subcutaneous nodules composed of granulation tissue, NOT bone) (commonly found on wrist, hands and fingers)
- 33. **Guards in chAinmail**: chronic systemic inflammation in RA can lead to AA (secondary) amyloidosis
- 34. **Settling red chimes**: systemic inflammation in RA causes elevation of inflammatory markers such as ESR (erythrocyte sedimentation rate)
- 35. **Magic CaRPet**: systemic inflammation in RA causes elevation of inflammatory markers such as CRP (C-reactive protein)

Musculoskeletal & Derm 2.3 - Gout & Pseudogout



- 1. **Uric acid knitting needles**: GOUT (monoarticular arthritis) (develops when needle-shaped URIC ACID CRYSTALS precipitate in joints)
- Red scarf: high SERUM uric acid levels (hyperURICEMIA) are necessary for development of gout (although many people with high levels never develop it)
- 3. **Pentagon-hexagon earrings**: breakdown of PURINES in the liver is the the major source of uric acid
- 4. Yellow needles & yarn retained in kidney bag: hyperURICEMIA is most commonly caused by UNDEREXCRETION of uric acid by the kidneys
- 5. **IDIOT**: UNDEREXCRETION of uric acid by the kidneys is usually idiopathic
- Two shrunken kidney heads: chronic KIDNEY disease can lead to gout (dysfunctional kidneys UNDEREXCRETE uric acid → hyperURICEMIA)
- 7. **Broken "PURE SALVATION" & "HiGh PRiesT"**: Lesch-Nyhan syndrome (inborn error of metabolism) (caused by mutations in HGPRT within Purine Salvage pathway → overproduction of uric acid) (causes gout, renal dysfunction, mental retardation, self-mutilation)
- 8. Yellow needle in ""HiGh PRiesT painting": Lesch-Nyhan syndrome can lead to gout (OVERproduction of uric acid \rightarrow hyperURICEMIA)
- 9. **Purine shards from shattered glass window**: Tumor Lysis Syndrome can cause gout (massive cell lysis \rightarrow PURINES released into bloodstream \rightarrow OVERproduction of uric acid \rightarrow hyperURICEMIA)
- 10. **Crab & B-cell antibody archers**: Tumor Lysis Syndrome most commonly occurs after chemotherapy for Acute Lymphoblastic Leukemia or Lymphoma)
- 11. **THIGHS**: THIAZIDE diuretics can precipitate gout (promote urate reabsorption in proximal tubule & deplete plasma volume → hyperURICEMIA)
- 12. **LOOPED cauldron handle**: LOOP diuretics can precipitate gout (promote urate reabsorption in proximal tubule & deplete plasma volume → hyperURICEMIA)
- 13. **DRY shrunken head** : DEHYDRATION can precipitate gout (hypovolemia → increased uric acid concentration)
- 14. Scalpels: SURGERY can precipitate gout
- 15. Bacterial lanterns: INFECTIONS can precipitate gout

- 16. **Mortar & Pestle**: DRUGS (probenecid, low-dose aspirin, paradoxical allopurinol effect) can precipitate gout
- 17. **Steak**: overconsumption of dietary sources HIGH in PURINES (red meat, organ meat, seafood) can precipitate gout
- 18. "XXX" Flask: ALCOHOL can precipitate gout (because competes with uric acid for excretion by kidney)
- 19. Large older man: male gender, obesity, and age >30 are risk factors for gout
- 20. **MacroCAGE with yellow needles**: elevated serum uric acid → macroPHAGES phagocytose urate crystals → release of inflammatory mediators
- 21. **First responders carrying cytoCOINS** : inflammatory mediators → recruitment of NEUTROPHILS → release more cytoKINES
- 22. Grimacing: ACUTE gout presents with PAIN at single joint
- 23. Red ball: ACUTE gout presents with swelling & redness at single joint
- 24. Warm candles: ACUTE gout presents with WARMTH at single joint
- 25. **Foot**: gout most commonly affects the MTP joint of great toe ("podagra")
- 26. **Sometime this month**: ACUTE gout usually resolves within days to weeks
- 27. **Yellow needle lodged into first responder**: on histology, acute gout displays URATE CRYSTALS phagocytosed within NEUTROPHILS
- 28. **First responders within fluid**: in acute gout, SYNOVIAL FLUID displays a high WBC count (predominately NEUTROPHILS)
- 29. **Persistently YELLOW urate needles in "NEGATIVE" pot**: Urate crystals display NEGATIVE BIREFRINGENCE
- 30. **Yellow parallel bi-re-FRAGRANCE sticks**: NEGATIVE BIREFRINGENCE indicates crystals appear YELLOW under PARALLEL polarized light

Musculoskeletal & Derm 2.3 - Gout & Pseudogout



- 31. **Grandfather clock with intermittent cuckoo**: gout is a CHRONIC disease with relapsing and remitting pattern
- 32. **Fiery PAN with layer of GRease**: chronic gout leads to formation of a PANNUS (layer of GRanulation tissue inside the joint capsule) (due to ongoing inflammation)
- 33. **TROPHY crystals on shawl & earrings**: Gout can be complicated by TOPHUS formation (urate crystals accumulate in subcutaneous tissue, cartilage, bone, ligaments → encased in granulation tissue to form a tophus) (most common in ear, adjacent to elbow or knee, Achilles' tendon)
- 34. **Kidney purse with yellow needles**: gout can be complicated by URATE NEPHROPATHY (urate crystals deposit in renal tubules and medullary interstitium → renal dysfunction) (additionally can precipitate to form uric acid stones)
- 35. **Necklace with CALCIFIED bone and PHOSPHATE fossiles**: CALCIUM PyroPHOSPHATE Crystal Deposition (CPPD) disease
- 36. **Hoodoo doll of gouty guest**: CPPD disease ("PSEUDOGOUT") can present with joint symptoms similar to gout
- 37. **Diamond needle in doll's knee**: CPPD disease most commonly affects the knee
- 38. Cartilaginous shark pin cushion: CPPD disease is caused by excess pyrophosphate production in the articular CARTILAGE → binds calcium to form CALCIUM PYROPHOSPHATE crystals within cartilage ("chondrocalcinosis")
- 39. IDIOT : CPPD disease is most commonly IDIOPATHIC
- 40. Oversized "Fe" weight: HEMOCHROMATOSIS increases the risk of CPPD disease
- 41. "PthD in Voodoo Sciences" : HYPERPARATHYROIDISM increases the risk of CPPD disease
- 42. Older lady: old age increases the risk of CPPP disease
- 43. **Torn dress**: a history of joint trauma or surgery increases the risk of CPPP disease

- 44. **Warm candles**: CPPP disease presents with WARMTH at single joint
- 45. **Red ball**: CPPP disease presents with swelling & redness at single joint
- 46. Exposed red knee : CPPP disease most commonly affects the knee
- 47. **Destroyed knee**: CPPP disease can progress to PYROPHOSPHATE ARTHROPATHY (joint destruction and arthritis
- 48. **Rhomboid incense holder**: CALCIUM PYROPHOSPHATE crystals display a RHOMBOID shape
- 49. **BLUE rhomboids with "+" markings**: Calcium Pyrophosphate crystals display POSITIVE BIREFRINGENCE
- 50. **Blue parallel bi-re-FRAGRANCE**: POSITIVE BIREFRINGENCE indicates crystals appear BLUE under PARALLEL polarized light
- 51. Linear White Rims (on skull and X-bone hat): in CPPD disease, X-rays display linear deposits of calcium within articular cartilage)

Musculoskeletal & Derm 3.1 - Muscular Dystrophies





- 1. "Du Seamus" clan: DUCHENNE muscular dystrophy (DMD)
- 2. "MacBecker" clan: BECKER muscular dystrophy (BMD)
- 3. Shy kid receding behind wall & "X" Scottish flag: DMD & BMD are X-linked recessive)
- 4. Long anchored "DESTROY" banner: DMD & BMD are caused by mutations in the DYSTROPHIN gene (largest protein-coding human gene) (cytoskeletal anchor protein in muscle cells)
- 5. Losing "E": dystrophin mutations are usually deletions of entire Exons
- 6. **Shifted FRAME**: DMD is caused by FRAMEshift mutations (a result of deletions that are not multiples of 3)
- 7. Crossed out "DESR": in DMD, there is NO production of functional dystrophin (because frameshift mutations lead to TRUNCATED or completely dysfunctional proteins)
- 8. Frame intact: in BMD, there is production of partially functional dystrophin (because deletions are multiples of 3)
- 9. **Muscled arms & heart design**: dystrophin encodes for an intracellular anchor protein in SKELETAL & CARDIAC myocytes
- 10. Curved wooden sign : cell membrane
- 11. Actin filament-esque support ropes : actin cytoskeleton (composed of actin filaments)
- 12. Falling "DESTROY" banner & cracked wooden sign: without dystrophin, myocytes are susceptible to mechanical injury
- 13. **Skull clasps**: in DMD & BMD, dysfunctional dystrophin → mechanical myocyte injury → myocyte NECROSIS → compensatory hypertrophy → replacement of muscle by adipose tissue and fibrosis)
- 14. Large spots next to small spots: in DMD & BMD, histology displays myocytes of various sizes (due to muscle regeneration in different phases)
- 15. **Central dots inside myocyte spots**: in DMD & BMD, myocytes display nuclei in the CENTER of the cells (rather than normally displaced along periphery)
- 16. Muscle tartan torn to reveal adiposity : in DMD & BMD, over time fibrosis and adipose tissue begin to replace muscle
- 17. **Blue spots WITH brown halo**: if dystrophin is PRESENT (even if in small amounts or low molecular weight [as in BMD]), immunoblotting for dystrophin will reveal a brown halo at the myocyte cell membrane
- 18. **Blue spots WITHOUT brown halo**: if dystrophin is ABSENT (as in DMD), immunoblotting for dystrophin will NOT reveal a brown halo at the myocyte cell membrane
- 19. Holding up "crisPy Chicken": in DMD & BMD, creatinine kinase (CK) is elevated (due to muscle breakdown)
- 20. Young kid with blue spots: DMD is more severe (than BMD) and presents early in life (by ages 2-3)
- 21. Kid with blue spots unable to catch up: DMD presents with muscle weakness early in life
- 22. **Waddling duck**: in DMD & BMD, muscle weakness begins in proximal lower extremities (including muscles of pelvic girdle and quadriceps → waddling gait)
- 23. **Oversized calves**: DMD (and later onset in BMD) presents with pseudohypertrophy of gastrocnemius (calf muscle) (starts with muscle hypertrophy to compensate for proximal muscle weakness → as disease progresses, this enlarged muscle is also replaced by adipose and fibrosis ["pseudohypertrophy"])

- 24. Arms propping up legs: in DMD (and late onset in BMD), patients rise to standing by propping themselves up by their arms, then using arms to push legs to standing ("Gower maneuver") (to compensate for proximal lower extremity weakness)
- 25. Adolescent with brown-halo spots: in BMD, symptoms develop in late childhood or adolescence
- 26. **Dilated heart sac race**: DMD & BMD can present with dilated cardiomyopathy (same mechanism as in skeletal myocytes) (absent/dysfunction dystrophin \rightarrow unstable cytoskeleton and cell membrane \rightarrow myocyte necrosis \rightarrow compensatory hypertrophy \rightarrow fibrosis)
- 27. Floppy heart balloon: DMD & BMD can present with heart failure (due progressive myocyte death and fibrosis)
- 28. **Shaking heart sac**: DMD & BMD can present with cardiac arrhythmias (due to progressive myocyte death and fibrosis)
- 29. "My Town": Myotonic Dystrophy (most common muscular dystrophy in adults of European descent)
- 30. Domino gate: Myotonic Dystrophy is autosomal dominant
- 31. DaMP rocK: dystrophia myotonica protein kinase (DMPK) gene
- 32. **CoTtaGe expansion**: Myotonic Dystrophy is caused by CTG trinucleotide repeat expansion in the DMPK gene
- 33. Family waiting in "anticipation": in Myotonic Dystrophy, the disease becomes more severe with each generation ("anticipation") (due to increasing numbers of trinucleotide repeats)
- 34. Population "50" : symptoms of Myotonic Dystrophy begin occurring in patients with > 50 repeats
- 35. Large spots next to small spots: in Myotonic Dystrophy, histology displays myocytes of various sizes (due to muscle regeneration in different phases) (similar to DMD & BMD)
- 36. **Central dots inside myocyte spots**: in Myotonic Dystrophy,, myocytes may display nuclei in the CENTER of the cells (rather than normally displaced along periphery) (similar to DMD & BDM)
- 37. **Thin weak arms**: Myotonic Dystrophy presents with muscle weakness and wasting of distal forearms & hands
- 38. Long narrow face with drooping mouth and ptosis: Myotonic Dystrophy presents with muscle weakness and wasting of the facial muscles \rightarrow long narrow face (temporal weakness), ptosis, mouth drooping
- 39. **Abnormal gate**: Myotonic Dystrophy presents with abnormal GAIT and foot drop (due to weakness of ankle dorsiflexors)
- 40. **Unable to let son go**: Myotonic Dystrophy presents with myotonia (slowed relaxation following normal muscle contraction) (manifests as "stiffness" or difficulty letting go of things)
- 41. Foggy monocle: Myotonic Dystrophy can present with cataracts
- 42. **Purse with tiny danglers**: Myotonic Dystrophy can present with testicular atrophy
- 43. Bald spot : Myotonic Dystrophy can present with frontal balding
- 44. **Shakey heart decoration**: Myotonic Dystrophy can present with cardiac arrhythmias or cardiomyopathy (similar to DMD & BMD)

Musculoskeletal & Derm 3.2 - Myasthenia Gravis & Lambert-Eaton Myasthenic Syndrome





- 1. Striated bricks: skeletal muscle
- 2. Electrical end plate: MOTOR-END PLATE on POSTSYNAPTIC surface of MUSCLE fiber
- 3. Round curb: PRESYNAPTIC NEURON
- 4. Between curb and outlets: SYNAPTIC CLEFT
- 5. Releasing Calci-Yum ice cream : action potential in the MOTOR NEURON \to opening of PRESYNAPTIC voltage-gated CALCIUM channels \to CALCIUM ions enter nerve terminal
- 6. "Pints & Quarts": in the MOTOR NEURON, most PRESYNAPTIC voltage-gated CALCIUM channels are P/Q type
- 7. Releasing Acetyl-cola bottles across curb: in the axon terminal of the MOTOR NEURON (PRESYNAPTIC), accumulation of CALCIUM → signals vesicles containing Acetylcholine (ACh) to migrate and fuse to membrane → release of ACh into SYNAPTIC CLEFT
- 8. Smoking at striated wall: at the POSTSYNAPTIC motor endplate, ACh binds to NICOTINIC ACh receptors
- 9. "Ion Channel news" : the ACh receptor is a LIGAND-gated ION channel (ACh binds receptor \rightarrow Na and Ca move into the myocyte & K moves out \rightarrow DEPOLARIZATION of myocyte \rightarrow action potential \rightarrow muscle contraction)
- 10. Acetyl-cola dumpster : residual ACh in the synaptic cleft is hydrolyzed by acetylCHOLINesterase
- 11. "GRAVIS" graffiti & gravestone end plate: MYASTHENIA GRAVIS (MG) (autoimmune disorder of neuromuscular junction \rightarrow muscle weakness)
- 12. Antibody keys scaring smoker at electrical end plate : MG is caused by IgG autoantibodies against the nicotinic ACh receptors of the motor end plate
- 13. **Overzealous "high-five"**: in MG, binding of IgG to nicotinic Ach receptors leads to activation of COMPLEMENT cascade → destruction and depletion of receptors → decreased neurotransmission → muscle weakness
- 14. **Turning over** : in MG, binding of IgG to nicotinic ACh receptors leads to INCREASED TURNOVER of the receptors \rightarrow decreased neurotransmission \rightarrow muscle weakness
- 15. Young woman : MG has bimodal onset (first peak \sim age 20, more common in women)
- 16. Older man : MG has bimodal onset (second peak ~ age 60, more common in men)
- 17. "Here LAy B8trice": MG is associated with HLA-B8 serotype (same HLA type associated with Graves disease)
- 18. Waxing & waning moon pattern : MG presents with fluctuating skeletal muscle weakness
- 19. **Tired of repeatedly snapping**: in MG, muscle weakness worsens with repetition (PRESYNAPTIC vesicles progressively become depleted of ACh → unable to release enough ACh to depolarize POSTSYNAPTIC membrane [requires a LOT of ACh due to IgG blockage of receptors])
- 20. "Closed for the night": MG presents with worsening symptoms towards end of the day (due to progressive depletion of ACh in presynaptic vesicles)
- 21. **Drooping eyelids**: MG can present with ptosis at the end of day (due to weakness of muscles keeping eyelids open)

- 22. Crosseyed streetlights: MG can present with diplopia (double vision) (due to weakness of extraocular muscles)
- 23. **Choking**: MG can present with dysarthria (difficulty speaking), dysphagia (difficulty swallowing), and difficulty chewing (impairment of muscles innervated by cranial nerves IX, X, XI, XII "bulbar symptoms")
- 24. Arms not raisable : progressive MG can present with difficulty raising arms (or less commonly legs) (due to weakness of proximal extremity muscles)
- 25. **Winded & blue face**: MYASTHENIC CRISIS presents with respiratory failure (requiring intubation) and sometimes bulbar weakness (→risk of aspiration)
- 26. Overgrown "THYME" : MG can present with thymic hyperplasia (and sometimes thymoma)
- 27. **Pulling out THYME**: in MG, thymectomy (removal of thymus) can improve symptoms (thymus facilitates production of antibody in MG)
- 28. **PHONE booth with TENSE phone wire** : edroPHONIUM (TENSILON) test can be used to diagnose MG
- 29. Acetyl-cola retained by slamming lid closed : edroPHONIUM is a SHORT-acting AcetylCholinesterase INHIBITOR \to DECREASED degradation of ACh \to INCREASED ACh in synapse
- 30. Surging with energy: in MG, administration of edroPHONIUM results in immediate increase in muscle strength (due to increased ACh in synapse)
- 31. **ICE on the face**: ICE pack test can be used to diagnose MG (cold ice placed over eyelid → INCREASES sensitivity of POSTSYNAPTIC ACh receptors & DECREASES breakdown of ACh by AcetylCholinesterase → increased eyelid muscle strength)
- 32. **STIGMA"** near retained Acetyl-cola : MG can be treated with pyridoSTIGMINE (AcetylCholinesterase Inhibitor \rightarrow Decreased degradation of Ach \rightarrow Increased Ach in synapse)
- 33. "LAMBS" graffiti : Lambert-Eaton Myasthenic Syndrome (LEMS) (autoimmune disorder of neuromuscular junction \rightarrow muscle weakness)
- 34. Antibody keys on Calci-Yum truck: LEMS is caused by IgG autoantibodies against PREsynaptic P/Q-type voltage-gated CALCIUM channels
- 35. Abandoned can of Acetyl-cola bottles: in LEMS, in the PRESYNAPTIC NEURON, IgG binds CALCIUM channels → Ach vesicles are not transported **Crab** shirt: in SOME cases, LEMS is induced by cancer cells (paraneoplastic syndrome)
- 36. Increasingly high jumps: in LEMS, repeated stimulation of motor neuron leads to LARGER muscle contraction (opposite of MG) (each stimulation of motor NEURON \rightarrow SMALL amount of CALCIUM escapes into nerve channel [despite antibodies] \rightarrow eventually enough accumulates to trigger ACh VESICLE fusion \rightarrow ACh released into synaptic cleft)
- 37. Striated pants torn at thigh: LEMS presents with SYMMETRIC muscle weakness of PROXIMAL LOWER EXTREMITIES
- 38. **Unable to stand**: LEMS presents with difficulty STANDING up or climbing stairs (due to proximal lower extremity weakness)
- 39. **Drying face mask**: LEMS can present with xerostomia (dry mouth) (due to autonomic dysfunction)
- 40. **Floppy hose**: LEMS can present with erectile dysfunction (due to autonomic dysfunction)
- 41. Small jail cell: paraneoplastic LEMS is usually caused by small cell lung cancer

Musculoskeletal & Derm 4.1 - Actinic Keratosis, Squamous Cell Carcinoma & Basal Cell Carcinoma





- 1. Actinic sand castle : actinic keratosis (solar keratosis)
- 2. Flat dead seaweed: stratum corneum (most superficial layer of skin composed of dead, denucleated keratinocytes)
- 3. Basal blue shells : stratum basale (basal layer of skin composed of keratinocyte stem cells)
- Sandy papillae: the epidermis and dermis are separated by the basement membrane with projections of dermis (papillae) extending into the epidermis
- 5. Actinic sun flag: UV light exposure is the most important risk factor for actinic keratosis (patients typically have strong history of sun exposure)
- 6. **Broken check point gate** : actinic keratosis (and SCC) is strongly associated with p53 tumor suppressor gene mutation (UV exposure (especially UVB) → DNA damage to p53 gene → unregulated keratinocyte proliferation)
- 7. **Atypical basal shells**: hyperplastic keratinocytes with cytologic atypia in the basal layer (WITHOUT invasion into dermis) are seen in actinic keratosis
- 8. Extra keratotic seaweed : actinic keratosis results in hyperkeratosis and thickening of the stratum corneum→ thickened, rough lesions
- 9. **Purple spots on seaweed**: parakeratosis (abnormal preservation of keratinocyte nuclei within the stratum corneum in actinic keratosis)
- 10. **Stretched out elastic swimwear**: UV damage to the dermis in actinic keratosis → thick, amorphous deposits of damaged elastic tissue (solar elastosis)
- 11. Actinic scaly shells: actinic keratosis lesions are small (<2cm) raised, rough papules with a whitish brown scaly appearance (hyperkeratosis)
- 12. Sandy spots on old man: lesions in actinic keratosis are found in older patients in sun exposed areas (face, scalp, ears, hands)
- 13. Pale beachgoer : hypopigmented skin has less melanin to protect from UV light→ increased risk of actinic keratosis
- 14. **Crab progressing toward actinic castle**: actinic keratosis has potential for malignant transformation to squamous cell carcinoma (half of squamous cell carcinoma develops from actinic keratosis)
- 15. Cancer crab castle: squamous cell carcinoma (SCC: malignant neoplasm of keratinocytes) like actinic keratosis is seen in older people, associated with CUMULATIVE UV exposure, fair skin, TP53 mutations
- 16. Digging hole by fire pit and stream : areas of chronic skin inflammation or scarring (burn scars, chronic ulcers, chronic draining sinus tracts) \rightarrow SCC (Marjolin's ulcer)
- 17. **Shedding sIcerotic snake** : lichen sclerosis (chronic epithelial inflammation forming smooth, white "parchment-like" skin lesions of the skin/vulva) \rightarrow SCC in rare cases
- 18. **Crutches kid between castles**: immunosuppression (HIV, steroids) is a risk factor for both squamous AND basal cell carcinoma
- 19. **PlAstic bottle washing up**: arsenic exposure (usually from contaminated water) is a risk factor for SCC
- 20. **Seaweed bow of "Castle In Situ"**: squamous carcinoma IN SITU (Bowen's disease) is SCC involving the full thickness of the epidermis WITHOUT extension into dermis (intact basement membrane)
- 21. **Abnormal shells infiltrating dermal sand**: spread of dysplastic keratinocytes THROUGH the basement membrane into the dermis→ INVASIVE squamous cell carcinoma

- 22. **Swirly pink pearl**: keratin pearls (concentric rings of eosinophilic keratin produced by abnormal squamous cells) are commonly seen in SCC
- 23. Pouty lower lip by castle : SCC most commonly develops in sun exposed areas of skin such as the head, neck, hands, and LOWER lip
- 24. **Red shells of "Castle In Situ"**: squamous carcinoma in situ (Bowen's disease) usually presents with well-defined, erythematous, scaly papules or plaques
- 25. **Ulcerated cancer crab**: invasive SCC often presents as a shallow ulcer surrounded by an erythematous, scaly, crusted plaque
- 26. **Hermit crab on nearby rocks** : regional lymph nodes are the most common site of metastasis in SCC (only 5% of cases)
- 27. "Sand Base": cutaneous basal cell carcinoma (cBCC) is the most common skin cancer worldwide (like Actinic keratosis and SCC, BCC development is linked to age, UV light exposure, and fair skin)
- 28. **Bad sunburn**: brief, intense sun exposure (sunburn) and childhood sun exposure are greater risk factors for BCC (rather than total sun exposure --> SCC)
- 29. Cancer fighting ribbon on patchy shorts: inactivating mutation of the Patched-1 (PTCH1) tumor suppressor gene is associated with BCC
- 30. **Sonic Hedgehog shirt** : inactivation of PTCH1 tumor suppressor gene→ ACTIVATION of Sonic Hedgehog regulatory pathway→ unchecked cell proliferation and development of BCC
- 31. **Eye patch and moustache in sand base**: BCC most commonly develops in sun exposed areas such as the lateral canthal folds and UPPER lip (opposed to LOWER lip in SCC)
- 32. **Nodular rocks in dermal sand and water** : nodular BCC features nodules and cords of palisading basophilic cells with a mucinous stroma in the dermis
- ${\bf 33. \ Shells \ lining \ edge}: nodules \ show \ nuclear \ palisading \ on \ edge$
- 34. **Pearly shells lining sand base**: nodular BCC presents with flesh-colored "pearly" nodules with rolled (raised) edges
- 35. **Red vines on sand base**: nodular BCC often contain telangiectasias (collections of dilated blood vessels forming fine red lines on the skin surface)
- 36. Dark sand base crater: BCC often have central areas of pigmented erosion or ulceration
- 37. **Basal shell rock under crusty boogie board**: superficial BCC features round buds of cancer cells in the dermal papillae and epidermis covered by a scaly, erythematous papule or plaque
- 38. **Bony driftwood by basal base**: BCC rarely metastasizes, but often invades surrounding structures such as cartilage and bone
- 39. **Kid hiding behind sign**: xeroderma pigmentosum is inherited in an autosomal recessive fashion
- 40. **Pigmented XP sunscreen**: patients with xeroderma pigmentosum are at increased risk for SCC, BCC, and melanoma
- 41. ["LiTTle Crab Beach": UV radiation damage to DNA can cause pyrimidine dimer formation (crosslinking of thymidine bases) that normally undergoes nucleotide excision repair (deficient in xeroderma pigmentosum)
- 42. "NO SCISSORS" sign : patients with xeroderma pigmentosum are unable to perform nucleotide excision repair→ extensive DNA damage with UV light exposure



- 1. **Top of window**: stratum CORNEUM of epidermis (layer of flattened dead cells)
- 2. **BASE of upper window**: stratum BASALE of epidermis (keratinocyte stem cell layer)
- 3. **Separating walkway**: BASEMENT MEMBRANE (separates epidermis above from dermis below)
- 4. **Ink spots at BASE of upper window**: MELANOCYTES (located in the stratum BASALE) (synthesize melanin)
- 5. **Depositing ink throughout upper window layer**: melanocytes pack melanin into melanosomes (small organelles) and transport them through dendrites → deposit melanin throughout epidermis
- 6. **Oversized irregular ink blot**: MELANOMA (neoplastic proliferation of melanocytes)
- 7. Ray of sunlight: UV exposure is the most significant environmental risk factor for melanoma (both UVA & UVB) (leads to DNA damage in skin cells)
- 8. **Sunburn**: intermittent periods of intense sun exposure (such as peeling sunburns) confer the greatest risk for melanoma
- 9. ${\it Kids}$: in particular, UV exposure during childhood is closely linked to development of melanoma
- 10. Pale with red hair, freckles, and light eyes: light skin, red hair, freckles, and light eyes predispose to melanoma
- 11. Larger brown spots: multiple nevi (normal moles) are associated with increased risk of melanoma (however, nevi are NOT precursor lesions for melanoma)
- 12. **Enlarged irregular ink blot**: ATYPICAL nevi (even a single lesion) are associated with increased risk of melanoma (display irregular borders, multiple colors, diameter > 5 mm)
- 13. Family: family history is a risk factor for melanoma
- 14. Cycle-DKN electric generator: FAMILIAL melanoma is commonly a result of mutations in CYCLIN-dependent kinase inhibitor 2A (CDKN2A)
- 15. Pink ribbon: CDKN2A encodes for the tumor suppressor p16
- 16. **One fallen pink ribbon**: CDKN2A-associated melanoma follows the "two-hit" hypothesis (one inherited germline mutation → cancer develops if a somatic mutation occurs during lifetime)
- 17. **Out of control CYCLE**: CDK2A mutations lead to unchecked progression through cell cycle (p16 normally inhibits cyclin-dependent kinases → regulates G1-S phase)

- 18. Applying XP sunblock on child: XERODERMA PIGMENTOSUM predisposes to melanoma at an early age (also squamous & basal cell carcinomas) (autosomal recessive FAMILIAL syndrome) (results in defective nucleotide excision repair)
- 19. "B. Fra": in melanoma, mutations in BRAF proto-oncogene are the most common ACQUIRED mutations
- 20. ${\bf MAP}$: BRAF mutations activate MAP-kinase and ERK signaling pathways \rightarrow unregulated cell cycle
- 21. "V600E": BRAF mutations typically involve a substitution of glutamic acid for valine (V600E mutation)
- 22. **VENOMOUS snake & nib fountain pen** : VEMURAFenib (targets mutated BRAF \rightarrow treats melanomas with BRAF mutations)
- 23. **Crutches**: immunosuppressed state increases the risk of melanoma (HIV, immunosuppressive medications)
- 24. **Transporting ink pots LATTERLY across walkway**: melanomas initially grow by spreading LATTERLY in the epidermis along DERMO-EPIDERMAL junction ("RADIAL growth")
- 25. Ink pot ascending to top of window : SUPERFICIAL SPREADING melanomas (most common melanoma subtype) (extend UPWARDS into superficial epidermis following radial growth phase \rightarrow forms "melanoma IN SITU"
- 26. Spots on back and legs: SUPERFICIAL SPREADING melanomas are most common on back and legs (sun-exposed areas)
- 27. **Flat grey, brown & black spots**: SUPERFICIAL SPREADING melanomas appear as flat irregular lesions with grey, brown, or black color



- 28. **Ink pot descending below walkway**: following horizontal growth phase, melanomas advance to VERTICAL growth phase by breaking THROUGH basement membrane into dermis
- 29. Crab elevator beneath walkway: MALIGNANT melanoma (cells have advanced through basement membrane into dermis)
- 30. **Depth gauge**: in vertically-growing melanomas, DEPTH of invasion (Breslow's thickness) is the most important predictor of metastasis risk (deeper = greater risk)
- 31. **Disorganized collection of LARGE ink pots**: in melanoma, LARGE neoplastic cells form poorly organized nests
- 32. Pot spilling DIVIDING ink : melanoma cells display numerous mitotic figures
- 33. **Ink pots with large purple lids & red nobs**: melanoma cells contain large nuclei and eosinophilic nucleoli
- 34. **100 dollar bill**: melanoma cells stain positive for S-100 (expressed by cells with neural crest origin
- 35. CREST shield: melanocytes derive from neural CREST cells
- 36. "A": melanomas display Asymmetry
- 37. "B": melanomas display irregular or notched Borders
- 38. "C": melanomas display Color variability within same lesion
- 39. "D": melanomas are large (Diameter ≥ 6 mm)
- 40. Evolving "E": melanomas change rapidly ("Evolution")
- 41. Vertical exploration rocket : NODULAR melanoma (skips radial growth phase \rightarrow rapidly progresses to VERTICAL growth)
- 42. Death skull: NODULAR melanomas have poor prognosis
- 43. **Dark nodular helmet**: NODULAR melanomas present with darkly pigmented nodules
- 44. Ink stain near LATERALLY moving ink pot : : LENTIGO maligna (displays LONG period of radial [LATERAL] growth)
- 45. **Older sun-damaged face**: LENTIGO maligna occurs in elderly with chronically sun-damaged skin

- 46. **Ink spots on palms & soles**: ACRAL LENTIGINOUS melanoma (presents with dark spots on palms or soles of feet (areas NOT exposed to sunlight)
- 47. **Darker skin**: ACRAL LENTIGINOUS melanomas are the most common melanoma subtype among dark-skinned individuals
- 48. **Nail marks**: ACRAL LENTIGINOUS subtype can present with SUBUNGUAL melanomas (dark STREAKS underneath fingernails or toenails) (grow from nail bed into nail plate)
- 49. **Death skull**: ACRAL LENTIGINOUS melanomas have a very poor prognosis
- 50. Round white lanterns : melanomas initially spread to regional LYMPH NODES \rightarrow then metastasize to distant organs
- 51. Ink blot on head : melanomas commonly metastasize to brain $% \left(1\right) =\left(1\right) \left(1$
- 52. Ink blot on chest : melanomas commonly metastasize to lungs

Neuro 1.1 - Epidural Hematoma, Subdural Hematoma, & Subarachnoid Hemorrhage





- 1. Cracks on car: skull fractures are very common with EPIdural hematoma and often occur at the pterion (thinnest portion of lateral skull)
- 2. **Spilling "Motor Madness" red drink**: EPIdural hematomas are caused by traumatic injury to middle meningeal artery (branch of external carotid artery), which lies just beneath the skull
- 3. Firmly secured DURA pocket : the dura is firmly affixed to suture lines, inhibiting blood from crossing→ expansion INWARD
- 4. Black cat with white lens patch : on NON-contrast CT, EPIdural hematomas will have a hyperdense (bright) BICONVEX or "lens" shape (due to dural suture lines)
- 5. **Shifty bent pole**: expansion of EPIdural hematomas INWARD can compress and displace brain parenchyma to the contralateral side (midline shift)
- 6. **High pressure head balloon**: expansion of EPIdural hematoma --> increased intracranial pressure
- 7. **Happy guy counting down**: because an EPIdural hematoma takes time to expand, patients often have a period of normal neurologic function immediately following injury (lucid interval)
- 8. **Blow to the head**: increased intracranial pressure due to intracranial hemorrhage often presents with severe headache
- 9. Nauseated from bumping: increased intracranial pressure due to intracranial hemorrhage often presents with nausea and vomiting
- 10. **Knocked out**: increased intracranial pressure due to intracranial hemorrhage often leads to altered mental status and syncope
- 11. **Bottom falling out after crash**: increased intracranial pressure can lead to uncal herniation (downward displacement of the medial temporal lobe through the tentorium)
- 12. **3 pronged grill and blown headlight**: uncal herniation can cause compression of the outer parasympathetic fibers of the oculomotor nerve→ ipsilateral pupillary dilation ("blown" pupil)
- 13. **Hole in cart shell**: acutely, burr holes can be drilled into the skull to temporarily relieve intracranial hypertension due to epidural hematoma
- 14. Blue drink between DURA pocket and spider: SUBdural hematomas involve hemorrhage into POTENTIAL space between the dura and arachnoid mater
- 15. **Broken blue belts**: SUBdural hematomas are caused by tearing of bridging VEINS that drain the cortex into the dural sinuses (usually by sudden acceleration-deceleration injuries)
- 16. **Baby rattler**: SUBdural hematomas are common in shaken baby syndrome (and other child abuse) due to thin-walled bridging veins
- 17. **Wet war vet shirt** : chronic anticoagulation with drugs like warfarin increase the risk for subdural hematoma following injury (especially in elderly)
- 18. **Shrunken brain hat** : cerebral atrophy (age, neurodegenerative disease, chronic alcoholism) increase the distance traveled by bridging veins→ increased tension→ higher risk of subdural hematoma
- 19. **Crescent shaped blue drink spill** : SUBdural hematomas are NOT contained by suture lines, allowing them to expand along the skull → crescent shape
- 20. Black cat with crescent white spot: on NON-contrast CT, SUBdural hematomas will have a hyperdense (bright) CRESCENT appearance

- 21. **Confusing, wincing crescent grandfather clock**: CHRONIC SUBdural hematoma causes the slow onset of neurologic symptoms including headache, seizures, and even cognitive decline
- 22. **Fibrous trees around crescent clock**: chronic subdural hematomas stimulate fibroblasts to form collagen around the hematoma, forming a membrane (organized subdural hematoma)
- 23. **Dark crescent moon** : chronic subdural hematomas appear hypodense (dark) on non-contrast CT due to breakdown of blood and proteins→ less dense fluid
- 24. White line of new moon: rebleeding or "acute on chronic" subdural bleeding will cause of new hyperdense (white) areas to appear on non-contrast CT
- 25. **Red CSF river under arachnid**: subarachnoid hemorrhage causes bleeding into the cerebrospinal fluid space between the arachnoid and pia mater
- 26. **Round red spider abdomen**: most subarachnoid hemorrhages are due to rupture of a saccular (berry) cerebral artery aneurysm
- 27. **Skeleton frightened by thunderclap**: rupture of a cerebral artery aneurysms often cause sudden "thunderclap" headaches that patients describe as the "worst headache of my life."
- 28. Flaming, vomiting zombie with neck shackle: subarachnoid hemorrhage may cause fever, nausea and vomiting, and meningismus (neck extensor spasm from meningeal irritation)
- 29. Hiding from flashing lights : subarachnoid hemorrhage may cause photophobia
- 30. **Big head balloon**: left untreated, subarachnoid hemorrhage can expand the subarachnoid space→ intracranial hypertension
- 31. **Thick white center of circular spider web**: on NON-contrast CT, subarachnoid shows hyperdense (white) material filling the subarachnoid space (especially near the circle of Willis)
- 32. **Dripping yellow syringe**: CSF following lumbar puncture in SAH will show an increased red blood cell count and observable xanthochromia (pinkish-yellow tint to CSF due to hemoglobin breakdown products)
- 33. **Squeezing vascular red sleeve**: hemoglobin breakdown products in the CSF following subarachnoid hemorrhage can cause endothelial irritation and vasospasm
- 34. Black chocolate syrup on brain ice cream: vasospasm following subarachnoid hemorrhage can cause ischemic stroke (typically 1 week following initial bleed)
- 35. ["Need Mo' Dippin" ice cream sauce : nimodipine (a dihydropyridine calcium channel blocker) is administered to prevent vasospasm following SAH
- 36. **Anterior round red mask**: 85% of cerebral aneurysms form in the anterior circle of Willis (especially at the anterior cerebral artery-anterior communicating artery junction (areas of turbulent blood flow))
- 37. **Woman pausing ride** : cerebral aneurysms are more frequent in postmenopausal women due to decreased estrogen→ decreased collagen production→ weakened vessel walls prone to aneurysm formation or rupture
- 38. Smoker: smoking is a risk factor for cerebral artery aneurysms
- $39. \ \textbf{Flask}: excessive \ alcohol \ intake \ is \ a \ risk \ factor \ for \ cerebral \ artery \ aneurysms$
- 40. **3-gilled shark with loose screws** : vascular Ehler-Danlos syndrome leads to defective type 3 collagen in vascular walls→ increased risk of cerebral aneurysm formation and rupture
- 41. **Man holding kidney shaped balloons**: autosomal dominant polycystic kidney disease (ADPKD) is associated with cerebral berry aneurysm formation

Neuro 1.2 - Ischemic & Hemorrhagic Stroke



- 1. Black paint stroke on head: ischemic stroke
- 2. **Bristle clot on head** : thrombotic stroke (most often due to thrombosis in an intracerebral vessel→ ischemic stroke)
- 3. Yellow glob of paint on head: thrombotic strokes are most often due to atheromatous plaque rupture/thrombosis in cerebral vessels
- 4. **Bird poop dropping onto head** : embolic strokes are caused by proximal emboli (usually thrombus) lodging in cerebral vessels→ ischemic stroke
- 5. Wavy line by embolic bird nest: conditions that promote thrombus formation (atrial fibrillation, hypercoagulable states) are risk factors for embolic stroke
- 6. **Blue brain in low fluid** : hypoperfusion or hypoxemia can impair oxygen delivery to the brain→ hypoxic stroke (global ischemia)
- 7. **Cold, blue cadaver** : hypoperfusion or hypoxemia can impair oxygen delivery to the ENTIRE brain (not focal) \rightarrow global hypoxic stroke
- 8. Crack leaking red fluid: hypotension (hemorrhage, hypovolemia) leading to decreased cerebral perfusion pressure is a common cause of hypoxic stroke (global ischemia)
- 9. Lightning tattoo on cadaver : shock (from sepsis, decreased cardiac output following MI, or spinal cord injury) causes decreased cerebral perfusion pressure→ global hypoxic stroke
- 10. **Magnifying glass focused on brain**: transient ischemic attacks (TIA) cause REVERSIBLE ischemia to parts of the brain leading to FOCAL neurological deficits (such as sensorimotor loss or aphasia)
- 11. **Quarter hour clock**: transient ischemic attacks (TIAs) normally last less than 30 minutes, with most resolving within 15 minutes
- 12. **Rubbing off black smudge**: transient ischemic attacks (TIA) involve REVERSIBLE ischemia WITHOUT INFARCTION or permanent brain damage
- 13. **Non-contrast black CAT** : NON-contrast head CT (to differentiate ischemic from hemorrhagic stroke) is the first step in evaluating acute stroke
- 14. White spot on CAT's head: blood in the brain from hemorrhagic stroke will show up as hyperdense (bright white) areas on non-contrast CT
- 15. Magnet above head : brain MRI is the diagnostic test of choice in acute ischemic stroke
- 16. **Weighted scale test**: diffusion WEIGHTED MRI can show areas of ischemia in the brain (bright white appearance) as early as 30 minutes following initial stroke onset
- 17. **Wedge shaped hat feather**: since vessels fan outward as they reach the cortex, blockage of proximal vessels in acute ischemic stroke causes a WEDGE shaped area of ischemia on imaging
- 18. **Red spots on portrait**: 12-24 hours following infarction, neurons develop vacuolated, eosinophilic cytoplasm as nuclei shrink and break apart (karyorrhexis) → appearance of "red neurons"

- 19. **EMTs entering through sundial door** : around 24 hours following infarction, neutrophils enter injured parenchyma→ inflammation, degradation, and phagocytosis of cellular debris
- 20. **Black necrotic liquid**: following brain infarction, neutrophils release digestive enzymes that break down dead tissue into a viscous liquid→ liquefactive necrosis
- 21. **Debris in macroCAGE**: around 3 days following infarction, macrophages and microglial cells (phagocytic cells of the CNS) phagocytize cellular debris and necrotic brain parenchyma
- 22. **Elevated hot air balloon head**: inflammation following brain infarction leads to cerebral edema→ intracranial hypertension if severe (as with large area infarctions)
- 23. Astro star flying machine: about a week following infarction, astrocytes proliferate to and provide structural support surrounding the area of liquefactive necrosis (reactive gliosis)
- 24. **Red string holding edges of astrostar machine**: astrocytic reactive gliosis promotes angiogenesis at the periphery of necrotic brain lesions following infarction
- 25. Water filled grandfather clock: liquefactive necrosis of infarcted brain tissue ultimately leads to formation of a fluid-filled cyst surrounded by a glial scar (as opposed to collagen-filled lesions formed in coagulative necrosis)
- 26. **Yellow gunk on middle of necklace**: large vessel thrombotic strokes are usually due to rupture or thrombosis of atherosclerotic plaques in the proximal Circle of Willis (most commonly in the middle cerebral artery)
- 27. **Yellow gunk on bottom of necklace**: large vessel thrombotic strokes secondary to atherosclerosis commonly occur in the vertebral arteries (near their origin from the subclavian arteries or intracranially where they meet to form the basilar artery
- 28. **Yellow gunk on bifurcated tree**: large vessel thrombotic strokes secondary to atherosclerosis commonly occur in the carotid bifurcation and internal carotid artery (main blood supply to Circle of Willis)
- 29. **Hand spasming around brush** : vasospasm during migraines can decrease cerebral perfusion or cause thrombosis→ large vessel ischemic stroke
- 30. **Burning red candles**: inflammation in intracerebral vessel vasculitis (giant cell arteritis, Takayasu arteritis) promotes thrombogenesis→ large vessel ischemic stroke
- 31. **Sickle palette knife**: sickling of erythrocytes and thrombus formation in sickle cell disease can cause large vessel ischemic stroke (especially in children)
- 32. **Lenticulostriate tree**: small vessel thrombotic strokes most commonly occur in the lenticulostriate arteries (small branches of the ACA and MCA that perfuse deep structure like the basal ganglia)

Neuro 1.2 - Ischemic & Hemorrhagic Stroke



- 33. Little lakes in painting: lenticulostriate artery thrombotic strokes occur in deep structures (basal ganglia, internal capsule, midbrain) and result in formation of small cysts (lacunar infarct)
- 34. **Geyser near lenticulostriate tree**: hypertension is the most important risk factor for lacunar infarcts
- 35. **Jar of candy**: poorly controlled diabetes mellitus is a risk factor for thrombotic stroke (especially lacunar infarcts)
- 36. **Scaly red paintbrush with pink gunk**: lipohyalinosis is caused by hypertension-induced endothelial damage→ leakage of plasma proteins into vessel walls→ hyaline thickening, arteriosclerosis, and lumen narrowing→ lacunar infarct
- 37. **Small yellow gobs on paint brushes**: hypertension and diabetes mellitus promote atherosclerosis→ microatheroma formation in small vessels of the brain→ lacunar infarct
- 38. Question mark brush cup: chronic accumulation of lacunar infarcts may the one of the causes of vascular dementia (second most common form of dementia following Alzheimer disease)
- 39. **Broken left lute heart string**: most emboli causing ischemic stroke come from the left heart (atrial fibrillation, mural thrombus following MI)
- 40. **Plant growth on valve** : valvular vegetations (as in infective endocarditis) can dislodge and travel into cerebral circulation→ embolic stroke
- 41. **Bird nest lower on red pipe**: embolic stroke can be caused by embolism or thrombus dislodgement from proximal atherosclerotic lesions of the aorta, carotid arteries, or proximal cerebral vessels
- 42. **Thrombotic bird nest in lung tree** : dislodged thrombus from DVT usually travels through the pulmonary arteries and into the lungs→ pulmonary embolism
- 43. **Bird carrying straw through patent oval hole**: with a patent foramen ovale (PFO), embolism from DVT can travel from the right atrium to the left atrium and into systemic and cerebral circulation (paradoxical embolism)→ embolic stroke
- 44. **Bird poop on painting**: the middle cerebral artery, due to its size and direct takeoff from the internal carotid artery, is the most commonly involved vessel in embolic stroke
- 45. **Multiple poop bombs on hat**: embolic strokes may affect multiple vascular territories due to multiple emboli produced by the same source
- 46. **Holding red paintbrush with 3 fingers extended**: tPA may be given in thrombotic and embolic stroke to dissolve thrombus, optimally within 3 hours of symptom onset
- 47. **Blocking red splash with 4 fingers of one hand and 5 on the other**: tPA may be given in thrombotic and embolic stroke to dissolve thrombus up to 4.5 hours following symptoms onset

- 48. **Red splash on kid's hat**: damage to the blood-brain barrier and free radical accumulation caused by ischemia in ischemic stroke can lead to hemorrhagic conversion upon restoration of blood flow (reperfusion injury)
- 49. **Yellow gunk on blue cadaver's head**: patients with pre-existing occlusive atherosclerotic lesions (carotid stenosis) or uncontrolled hypertension (higher baseline cerebral perfusion pressure) are particularly prone to hypoxic stroke
- 50. **Seahorse with triangular fins**: pyramidal cells of the hippocampus are the most vulnerable to hypoxia and are affected first in hypoxic stroke
- 51. **Branching coral**: watershed areas (areas where two arterial supplies connect via small anastomotic branches) are most vulnerable to hypoxic strokes
- 52. **Wedge-shaped frog feet**: watershed infarcts appear as bilateral, wedge-shaped ischemic areas in the anterior and posterior cerebrum
- 53. Red paint on head of portrait: hemorrhagic stroke (spontaneous intracerebral hemorrhage) accounts for only 20% of strokes
- 54. **Steaming angry**: chronic hypertension is the most important risk factor for hemorrhagic stroke
- 55. **Cerebral red amyloid chains**: cerebral amyloid angiopathy (caused by deposition of beta-amyloid in cerebral vessel walls) causes recurrent lobar and cerebellar hemorrhagic stroke (most commonly in elderly)]
- 56. **Red candles overhead**: inflammation in cerebral vessel vasculitis damages arteries→ intracerebral hemorrhage
- 57. **Star pattern on hat**: brain tumors (such as astrocytomas) can cause cerebral edema, inflammation, and intracerebral hemorrhage
- 58. **Scaly red brush**: chronic hypertension can lead to fibrinoid necrosis of cerebral vessels (especially small vessels)
- 59. **Pink gunk on cracked jar**: chronic hypertension can lead to hyaline arteriosclerosis of cerebral vessels (especially small vessels)
- 60. Cracked jar with scaly brush and pink gunk : chronic hypertension can cause fibrinoid necrosis and hyaline arteriosclerosis of cerebral vessels \rightarrow intracranial hemorrhage
- 61. **Sharp berry bush** : chronic hypertension→ fibrinoid necrosis and hyaline arteriosclerosis→ Charcot-Bouchard microaneurysm formation in small cerebral vessels (prone to rupture)
- 62. Red branches near sharp berry bush : Charcot-Bouchard microaneurysms are especially common in the lenticulostriate arteries (deep brain perforators originating from the ACA and MCA)
- 63. Red leaves of cerebellar tree: small vessels supplying deep cerebellar nuclei are prone to hypertensive vasculopathy and hemorrhagic stroke

Neuro 2.1 - Intracranial Hypertension (ICH)



- 1. ">XX": Intracranial hypertension (ICP >20mmHg)
- 2. Large ventricle helmet: hydrocephalus (increased CSF in ventricles) can cause intracranial hypertension
- 3. Rain filling brain hat : cerebral edema (intracellular or interstitial) can cause intracranial hypertension
- 4. Swollen cell basket : intracellular cerebral edema (fluid moves INTO cells from interstitium)→ intracranial hypertension
- Falling peanut bag : hyponatremia decreases extracellular osmolality→ water is drawn INTO brain cells→ cerebral edema
- 6. Hypoxic blue balloon : hypoxia → damage to brain cells (decreased Na+/K+ ATPase activity) → intracellular Na+ accumulation → water is drawn INTO cells → cerebral edema
- 7. Hugging white and grey: loss of grey-white differentiation is seen on imaging in cerebral edema
- 8. **Wet red ropes**: increased vessel permeability and fluid accumulation outside of brain cells (interstitium) → vasogenic (extracellular) intracranial hypertension
- 9. Cancer crab: brain tumors are a common cause of vasogenic intracranial hypertension (due to new leaky vessels)
- 10. **Red brain turban**: brain inflammation (e.g. meningitis/encephalitis) can cause vasogenic intracranial hypertension
- 11. Bird trauma : head trauma can cause vasogenic intracranial hypertension
- 12. **Electrocuted pilot**: headache and seizures are symptoms of intracranial hypertension
- 13. Vomiting balloon face : intracranial hypertension can cause vomiting
- Passed out: intracranial hypertension can cause altered mental status (e.g. confusion and coma)
- 15. **Swollen cylinder on retina balloon**: intracranial hypertension→ swelling of optic disc→ papilledema
- 16. Cross eyed : intracranial hypertension→ stretching of CN VI (palsy) → diplopia
- 17. Smooth area of basket: effacement of sulci can be seen on imaging in intracranial hypertension
- 18. High pressure steam, low heart watch, holding breath: HTN, bradycardia, irregular respirations = Cushing's reflex (seen in intracranial hypertension)
- 19. **Hole in stomach balloon**: intracranial hypertension can cause gastric (Cushing) ulcers (due to increased parasympathetic → increased gastric acid)
- 20. Herniating foot : brain herniation is a complication of intracranial hypertension
- 21. Middle balloon line: falx cerebri separates the left and right brain hemispheres
- 22. Lateral balloon line: tentorium cerebelli separates the cerebrum (above) from cerebellum (below)
- 23. Bottom of balloon : foramen magnum (at base of skull)
- 24. **Pirate swinging under falx line**: subfalcine herniation (cingulate gyrus herniates under falx cerebri)
- 25. Contralateral peg leg : subfalcine herniation \rightarrow compression of anterior cerebral artery (ACA) \rightarrow CONTRALATERAL lower extremity motor and sensory loss

- 26. **Hanging down middle**: transtentorial herniation (inferior occipital lobe and brainstem move down past the tentorium cerebelli)
- 27. **Dead and bloody** : transtentorial herniation \rightarrow shearing of basilar artery branches \rightarrow Duret hemorrhages (lethal)
- 28. **Hang down the side** : uncal herniation (medial portion of temporal lobe herniates past tentorium cerebelli)
- 29. **Dilated pupil telescope looking down and out** : uncal herniation→ compression of CN III→ mydriasis (due to unopposed sympathetic tone) and "down and out" (due to unopposed action of CN IV and VI)
- 30. **Black cloud on opposite side**: uncal herniation→ compression of posterior cerebral artery (PCA) → ipsilateral occipital lobe infarction→ CONTRALATERAL homonymous hemianopsia (with macular sparing)
- 31. Limp ipsilateral limbs : uncal herniation→ compression of CONTRALATERAL cerebral peduncle→ IPSILATERAL hemiparesis
- 32. **Dead man hanging from the bottom**: tonsillar herniation (cerebellar tonsils herniate into the foramen magnum)→ brainstem compression→ death
- 33. Fast breathing: hyperventilation can lower intracranial pressure (treat ICH)
- 34. **Tight red sleeve**: hyperventilation→ decreased PaCO2→ cerebral vasoconstriction→ decreased intracranial pressure (treat ICH)
- 35. Man with tall hat: mannitol causes osmotic diuresis to decreased cerebral edema and lower intracranial pressure
- 36. **Torn hat leaking water**: ventriculostomy can drain CSF and lower intracranial pressure (treat ICH)
- 37. "Idiot": idiopathic intracranial hypertension (IIH) (aka pseudotumor cerebri aka benign intracranial hypertension)
- 38. Obese woman : IIH occurs more often in obese women (ages 15-45)
- 39. Small balloon head: IIH is NOT associated with ventricular enlargement
- 40. Oral contraceptive pill design : OCPs increase risk of developing IIH
- 41. Big A: all-trans retinoic acid (used to treat APL) increases risk of developing IIH
- 42. Cycle propeller: Tetracyclines increase risk of developing IIH
- 43. White capped peaks: isotretinoin (vitamin A derivative used to treat acne) increases risk of developing IIH
- 44. Swollen cylinder on retina balloon : intracranial hypertension (due to IIH) o swelling of optic disco papilledema

- 47. String of tin cans: intracranial hypertension (due to IIH) → pulsatile tinnitus
- 48. **High pressure syringe**: in IIH, lumbar puncture will show increased opening pressure (due to elevated intracranial pressure)
- 49. Dark glasses: in IIH, untreated papilledema can lead to permanent vision loss
- Battery acid: acetazolamide can be used to treat IIH (reduces rate of CSF production)
- 51. Slit vents: optic nerve sheath fenestration (treat papilledema in IIH)

Neuro 2.2 - Hydrocephalus





- 1. Pipes pumping in water: choroid plexus is where CSF is made
- 2. Lateral lakes: lateral ventricles (located in each cerebral hemisphere)
- 3. Later lake passage: interventricular foramina (connect the lateral ventricles to the third ventricle)
- 4. Third central lake: third ventricle (located in the midbrain between the two thalami)
- 5. Straight river with silver fish: cerebral aqueduct (aqueduct of Sylvius) connects the third and fourth ventricles
- 6. Lower 4th diamond lake: fourth ventricle (diamond shaped) (located anterior to the cerebellum)
- 7. **Medial diamond lake outlet**: foramen of Magendie (connects fourth ventricle to subarachnoid space)
- 8. Lateral diamond lake outlet: foramina of Luschka (connect fourth ventricle to subarachnoid space)
- 9. **Spiderweb on drain pipe**: CSF returns to systemic circulation via arachnoid granulations (projections of arachnoid mater into the dural venous sinuses)
- 10. Large ventricle helmet: HYDROCEPHALUS (enlargement of the ventricles [ventriculomegaly] due to increased CSF volume)
- 11. Broken drainage pipe : dysfunction of the arachnoid granulations \rightarrow decreased CSF drainage \rightarrow COMMUNICATING hydrocephalus
- 12. Walkie-talkie and ventricle helmet: COMMUNICATING hydrocephalus
- 13. **Mohawk** : meningitis can cause fibrosis of the arachnoid granulations \rightarrow COMMUNICATING hydrocephalus
- 14. Fibrous plants around pipes : inflammation can cause FIBROSIS of the arachnoid granulations \rightarrow COMMUNICATING hydrocephalus
- 15. **Blood dripping from spider web**: subarachnoid hemorrhage can cause fibrosis of the arachnoid granulations → COMMUNICATING hydrocephalus
- 16. High raft pressure : CSF accumulation due to communicating hydrocephalus \rightarrow INTRACRANIAL HYPERTENSION
- 17. **Obstructed river**: obstruction of CSF flow out of ventricles → noncommunicating (OBSTRUCTIVE) hydrocephalus
- 18. Lost cell-phone : NONCOMMUNICATING (obstructive) hydrocephalus
- 19. 2 cherries : Chiari malformation (especially type II) can cause NONCOMMUNICATING hydrocephalus
- 20. **Tiki torches** : congenital ToRCH infections (e.g. toxoplasmosis, rubella, CMV) can cause scarring of the meninges→ NONCOMMUNICATING hydrocephalus
- 21. Cystic #3 buoy: colloid cyst forming in the roof of the 3rd ventricle can block the interventricular foramina— NONCOMMUNICATING hydrocephalus
- 22. Pine Trees Germinating : extra-gonadal germ cell tumors can arise in the pineal gland (pinealoma) \rightarrow compression of cerebral aqueduct \rightarrow NONCOMMUNICATING hydrocephalus
- 23. Cancer crab: tumors in the posterior fossa and 4th ventricle can cause NONCOMMUNICATING hydrocephalus
- 24. High raft pressure: CSF accumulation due to noncommunicating hydrocephalus— INTRACRANIAL HYPERTENSION

- 25. **High pressure head balloon**: hydrocephalus (communicating and noncommunicating) can cause INTRACRANIAL HYPERTENSION]
- $26. \ \, \textbf{Dilated hot air cylinder underneath retina balloon}: intracranial \ hypertension leads to papilledema (swollen optic disc)$
- 27. **ProTECTed area**: cerebral aqueduct runs along the TECTUM of the midbrain (affected in hydrocephalus)
- 28. CALL US: damage to the SUPERIOR COLLICULUS (in the tectum) leads to vertical gaze palsy (Parinaud's syndrome) (due to hydrocephalus, tumor, stroke)
- 29. **Setting sun**: Parinaud syndrome aka "setting-sun sign" is a vertical gaze palsy (unable to look upwards) caused by compression of the superior colliculus in the tectum
- 30. **Dilated pupils**: in Parinaud syndrome pupils DO NOT constrict well with light (bilateral light-near dissociation)
- 31. Accommodations available sign: in Parinaud syndrome pupils WILL constrict with accommodation (bilateral light-near dissociation)
- 32. Crying baby dropping the bottle: symptoms of intracranial hypertension in infants include irritability, poor feeding, and developmental delay
- 33. Large helmet on child: congenital hydrocephalus leads to macrocephaly and a bulging fontanelle
- 34. Crying baby dropping the bottle: symptoms of intracranial hypertension in infants include irritability, poor feeding, and developmental delay
- 35. Normal Pressure Pool: normal pressure hydrocephalus (NPH)
- 36. **Broken drain with spiderwebs**: NPH may be caused by impaired CSF absorption by the arachnoid granulations
- 37. Walkie-talkie: NPH may be a type of communicating hydrocephalus
- 38. Old man: NPH most commonly occurs in elderly people
- 39. Chronic grandfather clock: NPH is a chronic condition developing over years— ventricles slowly enlarge— hydrocephalus and NORMAL CSF pressure
- 40. Rafting for Idiots: NPH is often idiopathic
- 41. **Pulling on chin straps** : slow enlargement of ventricles→ stretching of nerve fibers around cerebral ventricles→ interrupts signal transduction]
- 42. Leg stretched out in boat: NPH causes gait instability (wide-based, "magnetic" gait) due to damage to the sacral motor fibers ("WOBBLY")
- 43. **Wet pants**: NPH causes urinary incontinence by disrupting communication between cortex and sacral micturition center ("WET")
- 44. "?" on book: NPH causes dementia and decreased executive functioning by stretching the periventricular limbic fibers) ("WACKY")
- 45. **Big ventricle helmet**: NPH is associated with ventriculomegaly (enlargement of the ventricles) (WITHOUT enlargement of the sulci) and normal CSF pressure
- 46. **EXCAVATOR truck**: hydrocephalus ex vacuo is ventricular enlargement with a proportionate increase in the sulci (due to loss of brain tissue with normal aging or neurodegenerative disease)
- 47. Wrinkly brain helmet : cerebral atrophy makes the ventricles appear enlarged → hydrocephalus ex vacuo
- 48. Large grooves : sulci are enlarged in hydrocephalus ex vacuo

Neuro 2.3 - Adult CNS Tumors



- Crab metasta-ships: in adults, METASTASES are the most common CNS neoplasms
- 2. Crab pins on lung lapels: lung cancer commonly metastasizes to the CNS
- 3. Shirt with crab logos: breast cancer commonly metastasizes to the CNS
- 4. Black splotches on crab coat : melanomas commonly metastasizes to the CNS
- 5. **Ceiling magnets** : MRI is useful for imaging of CNS tumors
- 6. **Metasta-ships in front of RING-enhancing planet** : on imaging, METASTASES to the CNS appear as multiple solid, nodular, RING-enhancing tumors
- 7. **Metasta-ships at GREY-WHITE junction**: METASTASES to the CNS typically present at the GREY-WHITE junction in the brain
- 8. **Skull logo**: METASTASES to the CNS typically display an area of central NECROSIS
- 9. Radiation symbol: exposure to ionizing radiation to the head (particularly in childhood) increases the risk of CNS tumors
- 10. **Family photo**: certain genetic syndromes increase the risk of CNS tumors (tuberous sclerosis, neurofibromatosis 2, von Hippel-Lindau syndrome, Turcot syndrome)
- 11. Map with highlighted cerebrum & dark cerebellum: in adults, CNS tumors are most commonly SUPRAtentorial (involving cerebral hemispheres) (in contrast, pediatric CNS tumors are commonly INFRAtentorial [cerebellum])
- 12. Clutching head: CNS tumors can cause headaches
- 13. Question mark design : CNS tumors can cause fatigue, memory problems, and personality changes
- 14. **Shaking**: CNS tumors can cause seizures (focal or generalized) (due to compression of surrounding brain parenchyma)
- 15. **Brain magnifying glass**: CNS tumors can cause focal deficits (sensory loss, blindness, paralysis, gait disturbances, ataxia)
- 16. Steam coming from cracked inflated helmet: CNS tumors can cause intracranial hypertension (due to direct mass effect OR obstruction of CSF flow) (presents with headache, nausea/vomiting, papilledema)
- 17. Overgrown ARACHNOID uniform: MENINGIOMA (tumor of meningeal cells originating from the ARACHNOID mater) (BENIGN, most common CNS tumor in adults)
- 18. **Middle-aged female**: MENINGIOMAS are most common in females aged 40-60
- 19. Round antenna attached to head: MENINGIOMAS are well-circumscribed ROUND tumors growing out from the dura (may remain attached to the dura via a dural "tail")

- 20. Parted hair: MENINGIOMAS are most common in the dural folds (falx cerebri, tentorium cerebelli, venous sinuses)
- 21. Whorled design: MENINGIOMAS are composed of spindle cells in a whorled pattern
- 22. **Pink laminated belt** : on histology, MENINGIOMAS display Psammoma bodies (eosinophilic laminated calcium deposits)
- 23. Bright stars: ASTROCYTOMAS (most common glioma in adults)
- 24. **Glion with "IV" logo & outside BLAST**: GLIOBLASTOMA MULTIFORME (GBM, "Glioblastoma", alternate term for grade IV [high-grade] astrocytoma)
- 25. Oversized crab weapon : GLIOBLASTOMA is the most common MALIGNANT primary CNS tumor in adults
- 26. **Older male crewmember**: GLIOBLASTOMA is slightly more common in men & typically presents in fourth to sixth decades of life
- 27. Oversized wrinkly forehead : GLIOBLASTOMA is most common in the FRONTAL & TEMPORAL lobes
- 28. **Butterfly pattern**: GLIOBLASTOMA can present as a "butterfly glioma" (due to growth into the corpus callosum and across the midline)
- 29. RINGED planet: on MRI, GLIOBLASTOMAS display thick peripheral RIM enhancement
- 30. **Bands of purple dots & skulls**: on histology, GLIOBLASTOMA displays small round purple cells that line up along bands of necrosis ("pseudopalisading necrosis")
- 31. **Red hair, beard, and moustache** : GLIOBLASTOMA displays prominent microvascular proliferation → can lead to hemorrhage
- 32. Galactic Federation of Allied Planets: GLIOBLASTOMAS & oligoDENDROgliomas stain positive for Glial Fibrillary Acidic Protein (GFAP)
- 33. **"SLOW" oligodendrocyte ship** : oligoDENDROgliomas (SLOW growing tumors, resemble oligodendrocytes)
- 34. **Middle aged officer**: oligoDENDROgliomas are most frequent at ages 40-50
- 35. **Visor over upper head** : oligoDENDROgliomas are most common in the FRONTAL & TEMPORAL lobes
- 36. Glass of milk: oligoDENDROgliomas are frequently CALCIFIED
- 37. **Purple fried egg**: on histology, oligoDENDROglioma cells display "fried egg" appearance (round nuclei with granular chromatin & generous cytoplasm)
- 38. **Thin red net pattern**: oligoDENDROgliomas display networks of thin, fragile anastomosing capillaries ("chicken-wire" appearance)

Neuro 2.3 - Adult CNS Tumors



- 39. "VHL" logo: von Hippel Lindau (VHL) (genetic cancer syndrome) (predisposes to CNS and non-CNS tumors)
- 40. **Domino pattern**: VHL is autosomal dominant
- 41. **Pink ribbon & "3" logo**: VHL is caused by deletion of the VHL tumor suppressor gene on chromosome 3
- 42. **One fallen pink ribbon**: VHL follows "two-hit hypothesis" (one inherited germline mutation, then a somatic mutation acquired during lifetime)
- 43. Curly red hair & nearby BLAST: VHL most commonly presents with HEMANGIOBLASTOMAS (benign vascular lesions, occur in multiple CNS sites)
- 44. Red curly bun & curls down back: HEMANGIOBLASTOMAS affect the cerebellum & spinal cord (INFRAtentorial lesions)
- 45. Red curl on sunglasses: HEMANGIOBLASTOMAS affect the retina
- 46. **Well-circumscribed bun**: HEMANGIOBLASTOMAS are well-circumscribed vascular lesions
- 47. Large cystic chair: HEMANGIOBLASTOMAS are surrounded by large fluid filled cysts (adds to compression of adjacent tissue)
- 48. **PASSing saucer**: HEMANGIOBLASTOMAS stain positive for periodic acid-Schiff (PAS)
- 49. **Blood spill**: HEMANGIOBLASTOMAS are prone to rupture and subsequent hemorrhage (due to thin vascular walls)
- 50. Falling reddish sunglasses : HEMANGIOMAS in retina can bleed \rightarrow retinal edema, traction, and detachment
- 51. RENASCAR crab jacket: VHL is associated with clear cell RENAL carcinoma
- 52. Colorful shaved ice with adrenal topping: VHL is associated with pheoCHROMOcytomas in adrenal medulla
- 53. Purple knotted wires coming from cave-like hole: cavernous hemanglOMAS (tumors common in CNS and liver, clusters of thin-walled, dilated blood vessels)
- 54. **Broken wire in pool of blood**: hemanglOMAS are very prone to hemorrhage (because vessels contain a single endothelial layer and thin adventitia [NO elastic fibers or smooth muscle])
- 55. **Smooth muscle hair band**: HEMANGIOBLASTOMAS have fairly normal capillary architecture (includes smooth muscle and elastic fibers) (in contrast to hemangIOMAS)

- 56. Chess board with brain design: PRIMARY CNS LYMPHOMA (neoplastic lymphocytes proliferate in the brain, instead of within lymphoid organ)
- 57. **Crutches**: an immunocompromised state is a significant risk factor for CNS lymphoma (most common CNS malignancy in this group)
- 58. "Epstein's Bar" : CNS lymphoma in immunosuppressed patients is almost always associated with EBV infection
- 59. **Oversized B cell antibody archer** : CNS lymphomas are diffuse LARGE B cell lymphomas
- 60. White astral pieces surrounding red enemies: CNS LYMPHOMAS tend to be multiple and accumulate around blood vessels
- 61. Skull piece : CNS LYMPHOMAS often display areas of necrosis
- 62. **RINGED planet holograph** : on MRI, CNS LYMPHOMAS demonstrate RING enhancement
- 63. **Bulging pituitary bag**: PITUITARY ADENOMAS (benign overgrowth of one pituitary cell type)
- 64. Oversized white headphones with segmented head band : SCHWANNOMAS (neoplasm of Schwann cells) (most common in peripheral nervous system, but can form in CNS)

Neuro 2.4 - Pediatric CNS Tumors



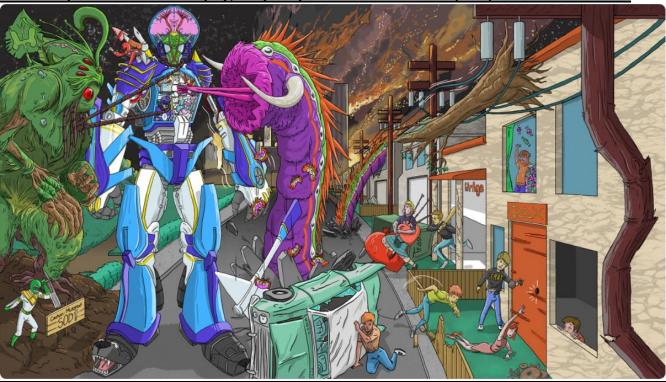


- Peeking out from under tent: most pediatric CNS tumors develop BELOW the tentorium cerebelli (INFRAtentorial) (in contrast, most adult CNS tumors are supratentorial)
- 2. Clutching head: CNS tumors can cause headaches
- 3. Shaking: CNS tumors can cause seizures
- 4. **Inflated head balloon**: CNS tumors can cause intracranial hypertension (presents with headache, nausea/vomiting, papilledema)
- 5. **Zombie prankster with green vomit**: INFRAtentorial CNS tumors can cause "cerebellar symptoms" (nausea/vomiting, gait problems, ataxia, impaired coordination)
- 6. Wet rafting helmet with oversized ventricles: INFRAtentorial tumors can cause obstructive HYDROCEPHALUS (due to obstruction of 4th ventricle)
- 7. Family time : certain genetic syndromes increase the risk of pediatric CNS tumors (tuberous sclerosis, neurofibromatosis 1)
- 8. Radiation symbol : exposure to ionizing radiation increases the risk of CNS tumors
- 9. Star pattern & tent with stick projections: PILOCYTIC ASTROCYTOMA (named for "hairlike" projections on microscopy) (most common pediatric CNS tumor, almost always occurs in children)
- 10. **Small doll**: PILOCYTIC ASTROCYTOMAS are grade 1 (benign, unlike astrocytomas in adults [higher grade & aggressive])
- 11. Benjamin Franklin doll : PILOCYTIC ASTROCYTOMAS frequently contain activating mutations in the BRAF gene \to activates abnormal cell growth
- 12. $\mbox{\bf Under the TENT}: \mbox{\rm PILOCYTIC ASTROCYTOMAS}$ are most common in the cerebellum (INFRAtentorial)
- 13. Well-circumscribed line : PILOCYTIC ASTROCYTOMAS are well-circumscribed tumors
- 14. Intermittent cystic puddles: PILOCYTIC ASTROCYTOMAS contain both solid and cystic components
- 15. **Stick projections**: PILOCYTIC ASTROCYTOMAS are composed of cells with long "hairlike" processes
- 16. Pink swirly pattern: in PILOCYTIC ASTROCYTOMAS, long processes of tumor cells contain Rosenthal fibers (EOSINOPHILIC CORSKSCREW-shaped glial filaments)
- 17. "GFAP" comic book: PILOCYTIC ASTROCYTOMAS stain positive for GFAP (because they are glial tumors)
- 18. Marshmallow BLAST near crab tent : MEDULLOBLASTOMA (most common pediatric MALIGNANT CNS tumor)
- 19. **Synapse fighting**: MEDULLOBLASTOMAS stain positive for SYNAPtophysin & NEURON specific enolase (because tumor originates from cells that are partially differentiated in the NEURONAL direction)
- 20. Under the tent: MEDULLOBLASTOMAS are most common in the cerebellum (INFRAtentorial)
- 21. **Diamond-shaped tent opening**: MEDULLOBLASTOMAS can extend into the 4th ventricle (diamond-shaped CSF collection)
- 22. Inflated head balloon : MEDULLOBLASTOMAS can cause intracranial hypertension (extension into 4th ventricle → obstructive hydrocephalus → ICH)

- 23. Multiple MICE: some MEDULLOBLASTOMAS contain MYC amplifications
- 24. **Soft BURNT marshmallows**: MEDULLOBLASTOMAS are soft friable tumors with areas of NECROSIS
- 25. Sheets of small BLUE marshmallows: MEDULLOBLASTOMAS are composed of sheets of small BLUE cells (due to hyperchromic nuclei and scant cytoplasm)
- 26. Ring of small BLUE marshmallows: on histology, MEDULLOBLASTOMAS display Homer-Wright ROSETTES (small BLUE cells surrounding acellular pink neuropil)
- 27. **DROPS of melted marshmallow**: MEDULLOBLASTOMAS may produce "DROP metastases" (CSF contains malignant cells, tumor nodules form along spinal cord)
- 28. **PEN outlining VENTRICLE drawing**: EPENDYMOMA (CNS tumor that arises from cells lining the VENTRICULAR system [ependymal cells])
- 29. **Diamond-shaped tent opening**: EPENDYMOMAS most commonly grow from the roof of the 4th ventricle (diamond-shaped CSF collection)
- 30. Inflated head balloon : EPENDYMOMAS can cause intracranial hypertension (extension into 4th ventricle \rightarrow obstructive hydrocephalus \rightarrow ICH)
- 31. Adult with PEN & white spinal stripe: in adults & neurofibromatosis type 2, EPENDYMOMAS occur along the SPINAL cord
- ${\bf 32.} \ \textbf{Well-circumscribed line}: \texttt{EPENDYMOMAS} \ are \ well-circumscribed \ tumors$
- 33. Intermittent cystic puddles: EPENDYMOMAS contain both solid and cystic components
- 34. Flowers with blue petals & extensions toward red center : EPENDYMOMAS display PERIVASCULAR pseudorosettes (cells form a circle around blood vessels → grow long extensions toward the center)
- 35. Flowers with blue petals & empty center : EPENDYMOMAS display ependymal rosettes (cancer cells cluster in an attempt to form a ventricle \rightarrow ring of cells around an empty lumen)
- 36. Cranial punching clown: CRANIOpharyngiomas (benign tumors arising from epithelial remnants of Rathke's pouch [oral ectoderm that ultimately forms anterior pituitary] (most common SUPRAtentorial pediatric tumor)
- 37. Cystic calcified face: CRANIOpharyngiomas display solid, cystic, and calcified components
- 38. "Germinating": primary CNS GERM cell tumors (extragonadal GERM cell tumors, result from abnormal migration of embryonal cells during development)
- 39. "PINE trees": the PINEAL gland is the most common location for CNS germ cell tumors (PINEALOMAS)
- 40. "Sea Man" action figure: primary CNS germ cell tumors are most commonly GERMINOMAS (similar to testicular SEMINOMAS)
- 41. Wet rafting helmet with oversized ventricles: PINEALOMAS can cause obstructive HYDROCEPHALUS (due to impingement on cerebral aqueduct)
- 42. **Inflated head balloon**: PINEALOMAS can cause intracranial hypertension (due to obstructive hydrocephalus)
- 43. Setting sun shirt: PINEALOMAS can lead to Parinaud syndrome (vertical gaze palsy syndrome, presents with "setting sun sign") (due to compression of the tectum & superior colliculi)

Neuro 3.1 - Spinal Muscular Atrophy, Amyotrophic Lateral Sclerosis (ALS) & Friedreich Ataxia





- 1. Surface of robot head : motor cortex (origin of upper motor neurons \rightarrow corticospinal tracts)
- 2. **Pyramids**: corticospinal (pyramidal) tracts start in the motor cortex and descend through medullary pyramids
- 3. Crossing wires: most of corticospinal tract fibers decussate (cross) at the medulla to form the lateral corticospinal tracts
- 4. Lateral wires beyond cross: lateral corticospinal tracts (upper motor neurons)
- 5. Lateral wire plugging into computer: corticospinal tract axons (upper motor neurons) synapse with lower motor neurons at the anterior horn
- 6. Anterior helmet projections: anterior horns of spinal cord (contain cell bodies of lower motor neurons)
- 7. **Break in upper wire**: upper motor neuron (UMN) lesion (causes muscle weakness, spastic paralysis, clasp-knife rigidity, and hyperreflexia)
- 8. Reflex hammer: UMN lesions cause HYPERreflexia
- 9. Clasp knife: UMN lesions cause spastic paralysis and clasp-knife rigidity
- 10. **Opening bite-binksy**: UMN lesions cause upgoing plantar reflexes (positive Babinski sign)
- 11. **Break in lower wire**: lower motor neuron (LMN) lesion (causes flaccid paralysis with hypotonia, hyporeflexia, muscle atrophy and fasciculations with downgoing plantar reflexes)
- 12. Thin arm: LMN lesions cause muscle atrophy
- 13. Losing grip: LMN lesions cause flaccid paralysis and hypotonia
- 14. Falling of reflex hammer: LMN lesions cause HYPOreflexia
- 15. Shaking arm: LMN lesions cause muscle fasciculations
- 16. **Spiny muscular space worm**: Spinal muscular atrophy (SMA) (congenital degeneration of anterior horns)
- 17. Spiny space worm attacking anterior horn synapse box : SMA is associated with degeneration of anterior horn cells→ LMN lesions (hypotonia, hyporeflexia, and muscle fasciculations)
- 18. **Spiny space worm bilateral horns**: SMA affects the anterior horns BILATERALLY (LMN symptoms present bilaterally)
- 19. **Baby Werdnig worms**: infantile SMA is known as Werdnig-Hoffman disease (presents before age 6 months)
- 20. Receding behind car: SMA (including Werdnig-Hoffman) has autosomal recessive inheritance
- 21. Polio flamingo : poliomyelitis (caused by the poliovirus) targets anterior horns \rightarrow LMN degeneration
- 22. Pink polio flamingo knight controlling one side : polio leads to ASYMMETRIC weakness/paralysis
- 23. Laterally sclerotic leaf: amyotrophic lateral sclerosis (ALS or Lou Gehrig's Disease) (progressive neurodegenerative disease of motor neurons)

- 24. Vines attacking upper head and lower controller of the robot : ALS causes UMN and LMN lesions
- 25. Laterally worn areas of leaf: ALS affects the bilateral LATERAL corticospinal tracts leading to atrophy and sclerosis
- 26. **Asymmetrically weak arm**: ALS causes ASYMMETRIC distal extremity weakness and atrophy
- 27. **Question mark**: ALS is associated with mild cognitive impairment (frontotemporal dementia)
- 28. Caution: Mutated SOD!: Superoxide dismutase type 1 (SOD1) gene mutations are frequently found in ALS
- 29. Razor weapon: riluzole improves survival in ALS (works by decreasing glutamate-induced excitotoxicity within neurons)
- 30. Phi Delta Chi frat house: Friedreich's ataxia
- 31. **Hiding frat brother**: Friedreich's ataxia is due to an autosomal RECESSIVE loss-of-function mutation in frataxin(FXN) gene on chromosome 9
- 32. **Line of GArAges**: expanded GAA repeat in frataxin gene causes Friedreich's ataxia
- 33. Ripped FRAT shirt : loss-of-function mutation in FXN gene \rightarrow decreased frataxin
- 34. **Broken iron knob off mitochondrial door**: frataxin is involved in iron homeostasis in the mitochondria
- 35. Accumulating rusty iron weights: decreased frataxin→ mitochondrial iron accumulation→ oxidative stress→ cell death (degeneration of multiple neural tracts and peripheral nerves)
- 36. **Tripping over gate**: Friedreich ataxia→ degeneration of spinocerebellar tracts and dentate nuclei→ gait ataxia
- 37. **Cracked columns** : Friedreich ataxia → degeneration of dorsal columns
- 38. Fallen tree on vibrating power lines : Friedreich ataxia → dorsal column sensory neuron loss→ impaired proprioception and vibration sensation
- 39. Weak legs : Friedreich ataxia \rightarrow lateral corticospinal tract degeneration \rightarrow lower extremity weakness
- 40. **High heels**: motor neuropathy in Friedreich ataxia causes foot abnormalities (e.g. pes cavus (high arched foot) and "hammer" toes)
- 41. **Twisted telephone pole**: kyphoscoliosis (abnormal curvature in the spine) is seen in Friedreich ataxia)
- 42. **Hypertrophic heart bag**: hypertrophic cardiomyopathy is seen in Friedreich ataxia (cardiac complications [e.g. arrhythmias and heart failure] are the most common cause of death)
- 43. **Elevated candy**: insulin resistance and diabetes is seen in Friedreich ataxia

Neuro 3.2 - Multiple Sclerosis





- 1. Losing myelinated fez hat: MULTIPLE SCLEROSIS (MS) (most common CNS demyelinating disorder) (caused by autoimmune destruction of oligodendrocytes)
- 2. White Olig with eye-like projections: oligodendrocyte (glial cell that forms myelin sheaths in the CNS) (contains cellular projections used to apply myelin around axons)
- 3. Young woman: MS most commonly affects women (with onset in early adulthood [age 20s-30]
- 4. "TelepHone poLice assistAnce, pull DooR 2 open" : the HLA-DR2 genotype increases susceptibility to MS
- 5. Viral light: viral infections may contribute to development of MS
- 6. **Great Britain highlighted on map**: MS is more common further from the equator
- 7. **Helper dropping cytoCOINS**: pathogenesis of MS may involve autoreactive helper T cells that inappropriately respond to antigens in myelin \rightarrow release inflammatory cytokines
- 8. **Blazing fire**: MS is characterized by formation of "active plaques" (localized areas of inflammation in white matter of brain or spinal cord)
- 9. MacroCAGES filled with white paper debris: on histology, active plaques contain abundant macroPHAGES phagocytosing myelin debris (as part of an inflammatory infiltrate)
- 10. **Pretending to be knight with T-shaped sword**: on histology, active plaques contain abundant CD8+ cytotoxic T cells (as part of an inflammatory infiltrate)
- 11. **Astral star pattern**: active plaques are surrounded by proliferating astrocytes (as a reaction to nerve tissue injury) ("active gliosis")
- 12. **One injured axonal arm**: in an active plaque, a small proportion of neuronal axons may be lost (although the majority remain uninjured)
- 13. **Dead Oligo robot with ASTRAL star pattern**: acute active plaques eventually progress to a chronic INACTIVE phase (axons now completely demyelinated, macrophages disappear, axons and oligodendrocytes are lost, ASTROCYTES fill lesion with a glial scar)
- 14. **Damaged area adjacent to ventricle hole**: plaques commonly form near the ventricles (although can form anywhere in the CNS)
- 15. **Disseminated in "Space"**: MULTIPLE sclerosis characteristically presents with neurologic signs and symptoms that cannot be explained by a single lesion
- 16. **Disseminated in "TIME"**: MS characteristically presents with a relapsing-remitting pattern of symptom progression
- 17. Hot & too tired to move: MS frequently presents with fatigue (particularly after hot showers or strenuous activity in hot environments)

- 18. Eye wire on fire: MS is the most common cause of OPTIC NEURITIS (inflammation of the optic nerve)
- 19. Covering eye in pain: OPTIC NEURITIS presents with unilateral eye pain (aggravated by ocular movements) & vision loss (primarily affecting central vision [central scotoma])
- 20. **Dilated eyes despite beam from stun gun**: OPTIC NEURITIS presents with a RELATIVE afferent pupillary defect (shining light in AFFECTED eye → bilateral pupils constrict LESS than normal (i.e. stay slightly DIIATED)
- 21. Swinging flashlight; in OPTIC NEURITIS, a RELATIVE afferent pupillary defect can be elicited on exam by shining light into normal eye then into affected eye \rightarrow subtle defect from the affected side becomes more obvious :
- 22. **Smacking robot eye with Madame Le Fleu" chair**: MS can present with INTERNUCLEAR opthalmoPLEGIA (lesion in Medial Longitudinal Fasciculus [MFL, coordinates horizontal eye movement]
- 23. **Injured eye unable to move inward**: in INTERNUCLEAR opthalmoPLEGIA (lesion in MFL), lesion in R side (for example) leads R eye to NOT ADDduct in response to ABDuction of L eye)
- 24. **Shaking L eye despite R eye injury**: INTERNUCLEAR opthalmoPLEGIA can cause horizontal nystagmus in unaffected eye
- 25. **Sensory blocking gloves**: MS commonly presents with sensory symptoms
- 26. Large reflex hammer: MS can present with hyperreflexia (upper motor neuron defect)
- 27. Clasping knife: MS can present with muscle spasticity ("clasp knife rigidity") (upper motor neuron defect)
- 28. **Electricity from robot's back** : MS can present with Lhermitte sign (neck flexion → electric shock sensation running down the spine)
- 29. **Leaking yellow and brown**: MS can present with bowel & bladder dysfunction (due to autonomic involvement)
- 30. **Shaking of pointed arm**: MS can present with intention tremors (due to cerebellar involvement)
- 31. **Scanning "SPEECH" podium**: MS can present with "scanning speech" (staccato disjointed sentences)
- 32. **Magnet**: MRI is used to diagnosed MS (reveals areas of demyelinated plaques)
- 33. Oligo clones with IgG attachments: in MS, protein electrophoresis of CSF reveals OLIGOCLONAL bands representing IgG antibodies (due to production of large amounts of IgG by multiple B cell lines)
- 34. **Punctured posterior pipe**: lumbar puncture (to collect CSF) is used to diagnose MS

Neuro 3.3 - Guillain-Barré & Charcot-Marie-Tooth

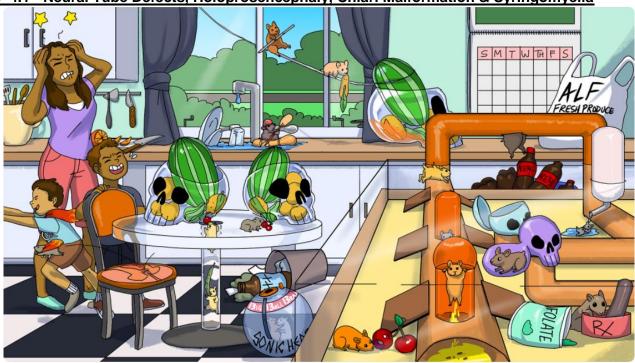


- 1. **Guillain the bear**: Guillain-Barre syndrome (GBS) (immune-mediated demyelinating disorder of PERIPHERAL nervous system) ("acute inflammatory demyelinating polyradiculoneuropathy")
- 2. **Myelin sleeping bags PERIPHERALLY around fire** : myelin sheaths in peripheral nervous system
- 3. **Mimicking**: GBS may be caused by an inappropriate immune response to a preceding infection (antigen of pathogen cross-reacts with peripheral nerve components [molecular mimicry])
- 4. **Unrolled torn myelin bag**: in GBS, the autoimmune response targets Schwann cells (responsible for producing myelin sheath of peripheral nerves)
- 5. Squiggly rod in CAMP fire: in GBS, the most common precipitating organism is CAMPYLObacter jejuni (S-shaped enteric gram negative bacteria) (causes bloody diarrhea in children) (spreads fecal/oral or via ingestion of undercooked meat)
- Viral lantern: GBS can be precipitated by viral infections (upper respiratory infections, HIV, EBV, CMV)
- 7. **HELPER bringing in the macroCAGES**: pathogenesis of GBS involves a CELLULAR immune response (including HELPER T cells recruiting macroPHAGES)
- 8. **Antibody skewers**: pathogenesis of GBS involves a HUMORAL immune response (may include autoantibody-mediated destruction of Schwann cells or myelin antigens)
- 9. Complimentary "high five": pathogenesis of GBS may involve local activation of the COMPLEMENT cascade (as a result of antibody binding)
- 10. **Swarm of little blue gnats**: on histology, GBS displays an endoneurial inflammatory infiltrate (abundant small cells with prominent blue nuclei) around peripheral myelin
- 11. **Unrolled myelin bag** : GBS presents with segmental demyelination throughout peripheral nerves \rightarrow impairs nerve conduction
- 12. **Unable to move tied shoes**: GBS characteristically presents with SYMMETRIC ASCENDING muscle paralysis
- 13. **Dropping reflex hammer**: GBS characteristically presents with decreased or absent deep tendon reflexes (DTRs)
- 14. **Bell collar** : GBS may present with bilateral facial paralysis (Bell's palsy) (if paralysis ascends high enough to involve CN VII)
- 15. **Blue face & trying to catch breath**: GBS may present with respiratory failure (due to paralysis of motor neurons innervating diaphragm & accessory respiratory muscles)
- 16. Alpine Campsite Association: in GBS, CSF displays "Albumino-Cytologic dissociation (high protein level BUT normal cell count) (due to increased capillary permeability at blood-nerve barrier)

- 17. **Marie's hounds-TOOTH outfit**: Charcot-Marie-Tooth (CMT) disease (hereditary motor sensory neuropathy) (group of disorders affecting development of PERIPHERAL nerve axons or their myelin sheath)
- 18. Domino purse: CMT is most commonly autosomal dominant
- 19. **Myelin sleeping bag & axonal firewood**: CMT is caused by defective production of proteins involved in the structure and function of peripheral nerves & myelin → demyelination and axonal dysfunction of peripheral nerves
- 20. **Too weak to step over sleeping bag**: CMT presents with progressive LOWER EXTREMITY WEAKNESS and atrophy (within first 2 decades of life)
- 21. Dropping foot : CMT presents with foot drop (lesions in common fibular nerve \to weakness in foot dorsiflexion)
- 22. **High heel**: CMT eventually leads to high-arched foot deformity ("pes cavus") (due to atrophy of nerves and muscles of the foot)
- 23. **Twisted trunk**: CMT eventually leads to thoracic kyphoscoliosis (curvature of the spine in both coronal and sagittal planes) (due to weakness of trunk muscles)
- 24. Sensory blocking stockings & gloves: CMT can present with sensory loss (particularly proprioception, vibration, and discriminate touch [senses normally carried on large, myelinated fibers])

Neuro 4.1 - Neural Tube Defects, Holoprosencephaly, Chiari Malformation & Syringomyelia





- 1. TUBE CITY: neural TUBES
- Tube at 4th calendar week: the NEURAL TUBE forms during the 4th week of fetal development (as a result of fusion of neural crests) (will eventually develop into brain and spinal cord)
- 3. Hole in the tube: NEURAL TUBE DEFECTS (NTDs) (occur as a result of failure of closure of anterior OR posterior neuropore) (most common congenital malformations of brain and spinal cord)
- 4. **Empty "Folate" bag**: FOLATE deficiency during very early pregnancy (prior to formation of neural tube at 4 weeks) can cause NTDs (recommend routine folate supplementation in women of childbearing age)
- 5. Mortar & pestle Rx bowel : drugs that impede folate metabolism (valproic acid, methotrexate, trimethoprim-sulfamethoxazole) can cause NTDs
- 6. **Elevated "Alf's Fresh Produce" bag** : ELEVATED alpha fetoprotein (AFP) levels in MATERNAL blood suggest a NTD in the fetus (open neuropore leaks CSF and proteins [including AFP] into amniotic sac \rightarrow AFP crosses placenta into maternal blood)
- 7. Overflowing trash can of Acetyl-Cola bottles: a NTD in a fetus presents with ELEVATED ACETYLcholinesterase in the amniotic fluid (open neuropore leaks CSF and proteins [including ACETYLcholinesterase] into amniotic fluid)
- 8. **Top of skull popping open**: ANENCEPHALY (complete lack of brain tissue & open skull) (NTD caused by failure of closure of ANTERIOR neuropore) (lethal)
- 9. Lying in a fluid puddle: ANENCEPHALY leads to polyhydramnios (excess amniotic fluid) during pregnancy (fetus without a brain is unable to swallow amniotic fluid → fluid builds up)
- 10. **Bubble budding off back of skull**: ENCEPHALOCELE (sac-like protrusion in the OCCIPITAL region) (contains herniated brain tissue or meninges) (NTD caused by failure of closure of ANTERIOR neuropore) (sometimes lethal)
- 11. Gaps in posterior tube : POSTERIOR neural tube defects (due to failure of closure of POSTERIOR neuropore)
- 12. Freely passing over UN-FUSED bridge: SPINA BIFIDA OCCULTA (non-fusion of posterior vertebral arch, usually in lumbar spine) (NO herniation of neural tissue) (POSTERIOR NTD)
- $13. \ \textbf{Tuff of hair}: SPINA \ BIFIDA \ OCCULTA \ often \ presents \ with a \ tuft \ of \ hair \ or \ skin \ dimple \ overlying \ the \ defect$
- 14. Partially caught in bridge CYST: MENINGOCELE (CSF filled cyst containing herniated meninges) (POSTERIOR NTD)
- 15. Fully caught in bridge CYST: MYELOmeningocele (CSF filled cyst containing herniated meninges AND lower spinal cord) (POSTERIOR NTD)
- 16. Peeing & pooping: MYELOmeningocele presents with neurological deficits at birth (starting from level of lesion downward) (motor, sensory, and/or bowel/bladder incontinence)
- $17. \ \textbf{Eating Chiari cherries}: \textbf{MYELOmening} occle is associated with CHIARI malformations$
- 18. Inseparable : HOLOPROSencephaly (left and right hemispheres fail to separate during development)
- 19. "'Sonic Hedgehog": HOLOPROSencephaly may be caused by mutations in the Sonic Hedgehog gene
- 20. "13ig 13all 13rand": HOLOPROSencephaly is associated with Trisomy 13 (Patau syndrome)
- 21. Beer bottle: HOLOPROSencephaly is associated with fetal alcohol syndrome

- 22. Cleft in ball: HOLOPROSencephaly can present with cleft lip or palate
- 23. One eye : severe HOLOPROSencephaly can present with cyclopia (a single eye)
- 24. Chiari cherry: Chiari malformations (congenital anomalies of the posterior fossa [cerebellum, brainstem, craniocervical junction] leading to downward displacement of cerebellum into spinal canal)
- 25. Wrinkly yellow fruit with midline leaf: CEREBELLUM (with cerebellar VERMIS running down the middle)
- 26. Oblong green fruit: MEDULLA OBLONGATA (part of brain stem)
- 27. Tonsillar cherry: round CEREBELLAR TONSIL (sits at the base of cerebellum)
- 28. Removing tonsillar cherry from posterior fossa bowl: Type I Chiari malformations (herniation of ONLY the cerebellar TONSILS down into spinal canal)
- $29. \ \textbf{Laughing kid}: Type \ I \ Chiari \ malformations \ are \ typically \ asymptomatic \ in \ childhood$
- 30. Holding head: Type I & 2 Chiari can present with headaches (due to meningeal irritation)
- 31. Dizzy stars: Type I & 2 Chiari may present with dizziness, nystagmus, and ataxia (due to cerebellar dysfunction)
- 32. Raised triangular hammer & clasp knife: type 1 & 2 Chiari may present with upper motor neuron dysfunction (hyperreflexia, clasp knife rigidity) (due to compression of corticospinal motor tracts in brainstem)
- 33. **STRING bean in central column**: SYRINGOMYELIA (cystic dilation of the central canal of spinal cord) (associated with Type I Chiari malformations)
- 34. Removing 2 tonsillar cherries, medullary green fruit, and vermis leafl: type II Chiari malformations ("Arnold-Chiari") (herniation of MEDULLA, cerebellar TONSILS, and VERMIS down into spinal canal)
- 35. Water accumulating from sink obstruction: Type II Chiari can present with NON-communicating (obstructive) HYDROCEPHALUS (downward displacement of cerebellar structures \rightarrow obstructs CSF flow through foramen magnum
- 36. WALKING on tight rope: Dandy-Walker syndrome (congenital brain malformation) (triad of agenesis of cerebellar vermis, cystic dilatation of 4th ventricle, and enlarged posterior fossa)
- 37. Stolen vermis leaf : Dandy-Walker is characterized by AGENESIS of cerebellar VERMIS
- ${\bf 38. \ Plentiful \ juicy \ CSF: Dandy-Walker \ is \ characterized \ by \ CYSTIC \ DILATION \ of \ 4TH} \ ventricle$
- 39. Enlarged back of fruit bowl : Dandy-Walker is characterized by ENLARGEMENT of POSTERIOR FOSSA
- 40. Water accumulating from sink obstruction: Dandy-Walker presents with NON-communicating (obstructive) HYDROCEPHALUS (due to agenesis of foramina of Luschka and Magendie)
- 41. Cape at lower neck: in SYRINGOMYELIA, dilation of central canal is most common in lower cervical cord (C8-T1)
- 42. Anterior white ties: in SYRINGOMYELIA, dilation of central canal compresses the ANTERIOR WHITE COMMISSURE
- ${\bf 43.}$ Jumping off central anterior cord : the ANTERIOR WHITE COMMISSURE is just anterior to central canal
- 44. Capes with red hot flames: SYRINGOMYELIA presents with loss of bilateral PAIN and TEMPERATURE sensation in upper extremities ("CAPE-like" distribution) (because pain and temperature fibers cross at anterior white commissure)

Neuro 4.2 - Sturge-Weber & Tuberous Sclerosis



- 1. "SouthWest" train: STURGE WEBER syndrome (SWS) (characterized by capillary-venous malformations in brain, eyes, and skin) (a neurocutaneous disorder)
- 2. Activated "coGNAQ" bottle : SWS is caused by an ACTIVATING mutation in GNAQ gene (encodes G alpha q signaling protein \rightarrow activates phospholipase C \rightarrow activates intracellular IP3-DAG pathway)
- 3. **Mosaic lamp**: SWS is characterized by MOSAICISM (some cells in the body carry the GNAQ mutation and some do not) (because mutation occurs in embryo AFTER fertilization → congenital SPORADIC disease [NOT inherited])
- 4. **Growth of red & blue spindly vines**: in SWS, GNAC gene mutation leads to overgrowth of small vessels (specifically at capillary-venous interfaces)
- 5. **Dark red wine splashed on face**: SWS characteristically presents with a NEVUS FLAMMEUS ("port wine stain", due to abnormal overgrowth of capillaries in skin)
- 6. Wine stain on forehead & upper eyelid: in SWS, NEVUS FLAMMEUS typically occurs in the distribution of the ophthalmic (V1) & maxillary (V2) branches of trigeminal nerve
- 7. **Red hat above wine stain**: SWS can present with LEPTOMENINGEAL ANGIOMAS (vascular malformations of the leptomeninges, typically IPSILATERAL to nevus flammeus)
- 8. Red weblike veil : in LEPTOMENINGEAL ANGIOMAS, capillary-venous malformations increase the vascularity of pia & arachnoid mater \rightarrow eventually also grow into subarachnoid space
- 9. **Skull accessory on hat**: LEPTOMENINGEAL ANGIOMAS can lead to brain ischemia and pressure necrosis (due to compression of brain parenchyma by overlying tortuous vessels)
- 10. **Shaking**: SWS can present with seizures (due to compression of brain by LEPTOMENINGEAL ANGIOMAS)
- 11. Too weak to hold purse on opposite side: in SWS, onset of seizures is associated with acute hemiparesis CONTRALATERAL to side of LEPTOMENINGEAL ANGIOMA
- 12. **Red lense over baby's eye**: SWS can present in infants with EPISCLERAL HEMANGIOMAS (capillary-venous malformations inside eye → can lead to increased intraocular pressure → early-onset glaucoma)
- 13. **Steaming large eyeball tea kettle**: if EPISCLERAL HEMANGIOMAS develop in utero, the increased intraocular pressure can lead to eyeball enlargement (buphthalmos)
- 14. Upside down "MENU": SWS typically presents with intellectual disability
- 15. **Snow on train tracks**: in SWS, X-ray or CT scan may reveal calcification of LEPTOMENINGEAL ANGIOMAS in "tram-track" pattern

- 16. Eating sclerotic tubers: TUBEROUS SCLEROSIS complex (TSC) (neurocutaneous disorder)
- 17. **Many HAMMERS**: TSC is characterized by formation of benign HAMARTOMAS throughout body (brain, eyes, heart, lungs, liver, kidneys, skin)
- 18. Domino lamp: TSC is autosomal dominant
- 19. **Pink ribbon logo**: TSC is caused by mutation in EITHER the TSC1 gene on chromosome 9 OR the TSC2 gene on chromosome 16 (both tumor suppressors)
- 20. Single broken potato chip: mutated TSC1 gene (encodes HAMARTIN)
- 21. Pair of broken potato chips: mutated TSC2 gene (encodes TUBERIN)
- 22. **Out-of-control cell cycle platter** : in TSC, mutations in TSC1 or TSC2 \rightarrow dysfunctional hamartin/tuberin complex \rightarrow constitutively active Ras pathway \rightarrow inappropriate activation of cell cycle
- 23. **Potato chips on brain platter**: TSC can present with GLIONEURONAL hamartomas ("cortical tubers", most common in cerebral cortex)
- 24. Potato chips lining brain platter: TSC can present with SUBEPENDYMAL nodules (CNS hamartomas that line the ventricular system)
- 25. **Helmet with CNS ventricle pattern**: in TSC, CNS hamartomas can cause CSF blockages (non-communicating hydrocephalus)
- 26. **Shaking kid**: TSC can present with seizures (as early as infancy, due to CNS hamartomas)
- 27. Upside down "MENU": TSC typically presents with intellectual disability
- 28. Adolescent with freckled cheeks: TSC can present with ANGIOFIBROMAS on malar regions of face (also called fibroadenomas or adenoma sebaceum) (begin in late childhood/adolescence with brown spots → grow larger, darker, and nodular over time)
- 29. **Skin decorated with thin chips**: TSC presents with ASH-LEAF spots (elliptical hypopigmented macules anywhere on skin, may be seen at birth)
- 30. **Salami patch on kid's back**: TSC can present with SHAGREEN PATCHES (plaques of dimpled, leathery skin, most common on lower back)
- 31. **Heart platter with red meat slices**: TSC can present with CARDIAC RHABDOMYOMAS (benign tumors detectable by prenatal ultrasound, spontaneously regress with age) (in infants, impingement on mitral valve may cause regurgitation)
- 32. Fibrotic lung bush : TSC can present with diffuse interstitial fibrosis \rightarrow can lead to dyspnea or pneumothorax
- 33. Kidney platter with vessely red veggies, meat slices, and fatty sauce: TSC can present with RENAL ANGIOmyoLIPOMAS (benign kidney tumors)
- 34. **Crab near kidney platter**: in TSC, RENAL ANGIOmyoLIPOMAS can undergo malignant transformation

Neuro 4.3 - Neurofibromatosis



- 1. "New Flavor cafe": Neurofibromatosis (NF) types 1 & 2 (neurocutaneous syndromes)
- 2. Counter "1": NF1 (most common form of NF)
- 3. "Von Recklinghausen 17" with pink ribbon logo: NF1 ("von Recklinghausen disease") is caused by a mutation in the NF1 tumor suppressor gene on chromosome 17
- 4. Domino sign: NF1 is autosomal dominant
- 5. "100% Peruvian": NF1 has 100% Penetrance (ALL individuals with the mutation [genotype] will have some manifestations of disease [phenotype])
- 6. "New Flavor" BROOM: NF1 encodes neuroFIBROMIN (GTPase that inhibits Ras signalling pathway)
- 7. **Broken BROOM & active RATS** : in NF1, mutated neuroFIBROMIN leads to constitutively ACTIVE RAS → uninhibited cell cycle activation
- 8. Coffee stains on tan apron: NF1 presents with CAFE-AU-LAIT SPOTS (flat, hyperpigmented macules) in the first year of life
- 9. **Brown spots near underarms**: NF1 presents with FRECKLING in skin folds (particularly axillary and inguinal regions) in childhood
- 10. **Eye pattern of "DeLisch Mocha"**: NF1 presents with LISCH NODULES (raised tan-colored hamartomas in the iris of the eyes) in later childhood
- 11. **Swirl in espresso foam**: NF1 presents with NEUROFIBROMAS (benign peripheral nerve sheath tumors composed of Schwann cells [which form a SWIRL of myelin around axons]) in adulthood
- 12. Foam bits on trunk: in NF1, NEUROFIBROMAS are usually CUTANEOUS (present as soft, fleshy, sessile or pedunculated growths) (mostly on trunk and neck)
- 13. "Spresso 100" with Crest logo: in NF1, CUTANEOUS NEUROFIBROMAS stain positive with S100 (indicates neural crest origin)
- 14. **Bulge in wires leading to eyeball lights**: NF1 can present with OPTIC GLIOMAS (tumors that form anywhere along anterior visual pathway [optic nerves, chiasm, nerve tracts])
- 15. Starry ceiling: OPTIC GLIOMAS are low grade, benign astrocytomas
- 16. **Pink swirls**: OPTIC GLIOMAS are composed of astrocytes with eosinophilic inclusion bodies (Rosenthal fibers)
- 17. **Hats with astral star logo**: NF1 can present with other CNS TUMORS (meningiomas, astrocytomas, other gliomas)
- 18. "Rainbow Frappe" with adrenal topping: NF1 can present with pheoCHROMOcytomas in the adrenal medulla

- 19. **Twisted broken bone broom**: NF1 can present with bony abnormalities (long bone dysplasia and fractures → leads to congenital PSEUDARTHROSIS [false joints that form when a long bone fracture does heal correctly]) (also SCOLIOSIS)
- 20. Counter "2": NF2
- 21. "22" steam : NF2 is caused by a mutation in the NF2 gene on chromosome 22
- 22. **MERLIN with pink hat ribbon** : the NF2 gene encodes for the tumor suppressor protein MERLIN
- 23. **Bilateral large white headphones with spiral design**: NF2 presents with bilateral VESTIBULAR SCHWANNOMAS (ACOUSTIC NEUROMAS, benign Schwann cell tumors, arise from vestibular portion of CN VIII)
- 24. **Unsuccessfully balancing cups**: VESTIBULAR SCHWANNOMAS can lead to vertigo and nystagmus (due to impingement of vestibular portion of CN VIII)
- 25. **Covered ears**: VESTIBULAR SCHWANNOMAS can lead to sensorineural hearing loss (due to impingement of cochlear portion of CNVIII)
- 26. **TIN tip jar**: VESTIBULAR SCHWANNOMAS can lead to TINnitus (due to impingement of cochlear portion of CNVIII)
- 27. Covered face & loose jaw at "V" table: VESTIBULAR SCHWANNOMAS can lead to loss of facial sensation or masseter weakness (due to impingement of trigeminal nerve [CN V])
- 28. **Droopy face & senseless tongue at "VII" table**: VESTIBULAR SCHWANNOMAS can lead to facial muscle weakness and loss of taste to anterior tongue (due to impingement of facial nerve [CN VII])
- 29. Encapsulated "ANTONI FRAPPE" with alternating dark & light pink layers: VESTIBULAR SCHWANNOMAS are ENCAPSULATED tumors of neoplastic Schwann cells (with alternating areas of hypercellularity [Antoni A] & hypocellularity [Antoni B]
- 30. **Register with "\$100" & Crest logo**: VESTIBULAR SCHWANNOMAS stain positive for S100 (indicates neural crest origin)
- 31. "DURA" beanie with 2 puff balls: NF2 can present with multiple MENINGIOMAS (benign growths of the dura)
- 32. Foggy glasses: NF2 often presents with CATARACTS
- 33. **Bulge in spinal wire**: NF2 often presents with SPINAL TUMORS → leads to motor dysfunction (spinal schwannomas, spinal meningiomas, and ependymomas)

Neuro 5.1 - Alzheimer Disease & Dementia





- 1. Scratching head in confusion: dementia is a CHRONIC neurocognitive disorder causing PROGRESSIVE decline in at least one area of executive cognitive ability (MEMORY, speech, attention) with CLEAR consciousness (as opposed to acute alteration of cognition in delirium)
- 2. **Old King Alzheimer**: age is the most important risk factor for Alzheimer disease (50% over 85 are affected)
- 3. **DOWNtown center by young boy**: Down syndrome is associated with early-onset Alzheimer disease (often occurring be 40)
- 4. **Young man with jeweled crown**: amyloid precursor protein (APP), the precursor to AMYLOID BETA, is encoded on chromosome 21→ trisomy 21 (Down syndrome) may increase APP production→ early onset Alzheimer disease
- 5. **Pre-king prince #1**: mutations in presenilin 1 (chromosome 14) may promote the production of amyloid beta→ early onset Alzheimer disease
- 6. **Pre-king prince #2**: mutation in presentilin 2 (chromosome 1) may promote the production of amyloid beta→ early onset Alzheimer disease
- 7. **E flags 4 the Alzheimer king**: homozygosity of the ApoE4 allele (chromosome 19) is a risk factor for late-onset Alzheimer disease (10x increased risk)
- 8. A beautiful crown : amyloid beta (derived from APP) are misfolded proteins that form aggregates called neuritic (senile) plaques in the brain→ Alzheimer disease
- Beta extracellular cape : accumulations of amyloid beta form
 EXTRAcellular plaques, especially in the medial temporal lobe (hippocampus, amygdala) → neuronal dysfunction and apoptosis → Alzheimer disease
- 10. Inner tau tunic: hyperphosphorylated tau protein (microtubule stabilizing protein) accumulates INTRAcellularly to form neurofibrillary tangles (especially in the medial temporal lobe) \rightarrow Alzheimer disease
- 11. **Small wrinkled hat**: neurodegeneration in Alzheimer disease leads to loss of cortical mass→ ventricles APPEAR enlarged on imaging (hydrocephalus ex vacuo)
- 12. **Hippocampal seahorses**: brain atrophy in AD is most prominent in the hippocampus and can be seen on MRI early in disease
- 13. No AcetylCola for the king: Alzheimer disease is associated with low acteylcholine levels
- 14. Cracked alChAmisT sign: decreased activity of choline acetyltransferase (ChAT) causes low acetylcholine levels in AD
- 15. **N Basalis plant**: the nucleus basalis of Meynert (responsible for memory and cognition) is especially prone to low acetylcholine levels and dysfunction in AD
- 16. "RECENT EVENTS" magazine: the predominant feature of AD is memory dysfunction (especially with recent events)
- 17. **King Alzheimer's executive planner**: executive cognitive dysfunction (difficulty planning or organizing) is a feature of AD

- 18. **Question map**: visuospatial impairment (a loss of sense of "self" in relation to surroundings) is a feature of AD (commonly presents as getting lost in familiar surroundings)
- 19. **Jester hat pointing to front and sides**: frontotemporal dementia causes preferential degeneration of the frontal and temporal lobes
- 20. **Autosomal domino pattern**: up to 25% of cases of frontotemporal dementia are inherited in an autosomal dominant fashion
- 21. **Young jester**: onset of frontotemporal dementia is typically in the fifth and sixth decades (opposed to the eighth in AD)
- 22. **Tau charm on jester hat**: the most common subtype of frontotemporal dementia is caused by tau protein inclusions in the brain (FTLD-tau)
- 23. **PICK** a shell to find the **TAU**: histologically, FTLD-tau (Pick disease) displays Pick bodies (hyperphosphorylated tau protein forming INTRAcellular inclusion bodies in neurons)
- 24. "Dancing Prankster 43": a common subtype of frontotemporal dementia is caused by accumulation of abnormally ubiquitinated TDP-43 RNA binding protein
- 25. Creepy smiling jester face : the BEHAVIORAL variant of FTD leads to early personality and behavior changes \rightarrow inappropriate behavior, apathy, hyperorality, and compulsivity
- 26. **Covering mouth**: the primary progressive aphasia variant of FTD causes early, progressive language dysfunction→ paucity of speech, repeated phrases, word-finding difficulty)
- 27. Frayed lateral spinal collar: FTD is associated with amyotrophic lateral sclerosis (ALS), as most patients with ALS show TDP-43 intracellular neuronal inclusions]
- 28. **King Lewy's alpha scepter**: DLB is caused by accumulation of alphasynuclein, which forms round, eosinophilic INTRACELLULAR inclusions
- 29. **Gears on King Lewy's tomb**: patients with DLB typically develop ParkinsonISM (bradykinesia, cogwheel rigidity, resting tremor) (Lewy bodies are seen in patients with Parkinson DISEASE as well)]
- 30. **Hallucination-like painting**: DLB commonly causes visual hallucinations early in the course of disease
- 31. **Falling down steps**: vascular dementia is characterized by STEPWISE decline in cognition (stable cognitive function punctuated by periods of acute, permanent decline)]
- 32. **Black stroke on painting's head**: accumulation of cerebrovascular events (strokes) are responsible for the stepwise decline in cognition in vascular dementia (infarcts of varying age seen on MRI)
- 33. **Evil white wizard**: cognitive decline and dementia may occur in patients with advanced HIV-AIDS (CD4 counts <200)
- 34. **Wizard's flame staff and cages**: HIV-associated dementia involves inflammatory activation of microglia and macrophages
- 35. MacroCAGED monster with multiple eyes: activated macrophages and microglial cells in HIV dementia release cytokines and cause inflammation→ formation of microglial nodules with necrosis and multinucleated giant cells

Neuro 5.2 - Creutzfeldt-Jakob Disease, Parkinson's Disease & Huntington's Disease



- 1. "MAD COW" tattoo: CJD is a spongiform encephalopathy that is TECHNICALLY an infectious disease (similar to eating beef tainted with bovine spongiform encephalopathy)
- 2. **Spiral lanyard by beta key**: CJD is caused by accumulation of misfolded prion protein (PrP), which undergoes transformation from the normal alpha-helix conformation (PrPc) to beta-pleated sheets (PrPsc)
- 3. Beta key next to beta key: PrPsc turns normal PrPc into more abnormal PrPsc, amplifying the process of prion accumulation
- 4. **Bubbly pink hat**: histologically, CJD causes vacuole formation in grey matter structures (such as the caudate nucleus and putamen) that develop into cysts (NO inflammation in prion disease)
- 5. **Spongy seat headrest**: vacuolization and cyst formation cause degradation of brain parenchyma giving it a "spongy" appearance
- 6. **Questionable antenna**: dementia progressing RAPIDLY over weeks to months is seen in CJD
- 7. **Startled arm jerking**: a classic motor finding in CJD is myoclonus (sudden, involuntary muscle contractions)
- 8. **Sharp wave auto decal** : electroencephalogram (EEG) changes seen with CJD include periodic sharp wave complexes
- 9. **Autosomal domino Hunting Town column**: Huntington disease is inherited in an autosomal dominant fashion with 100% penetrance
- 10. **Repeating CArGo pockets**: Huntington disease is caused by CAG trinucleotide repeat expansion on chromosome 4
- 11. "4": Huntington disease is caused by CAG trinucleotide repeat expansion on CHROMOSOME 4
- 12. ["Anticipate Great Savings" by wearing more pockets: copy number of CAG repeats increases through generations, leading to earlier and more severe disease (termed anticipation)
- 13. **CArGo pants father**: CAG copy number increases much more during spermatogenesis than oogenesis→ more severe disease if mutated allele is from father
- 14. ["Straight up, cod fishing, pull 'em in!" : HD causes atrophy of the STRIATUM, with the CAUDATE nucleus affected more than the PUTAMEN
- 15. **EXCAVATOR**: loss of brain parenchyma in HD can lead to the APPEARANCE of enlarged ventricles (hydrocephalus ex-vacuo) though they are normal
- 16. **Hiding in CAB-A**: loss of the inhibitory neurotransmitter GABA is the most common biochemical change seen in HD

- 17. **Middle-aged father**: Huntington disease typically presents between age 30-50
- 18. **Rapid dodging skills**: motor involvement in HD leads to chorea (rapid, involuntary or arrhythmic movement involving the face, trunk, and extremities)
- 19. **Angry clown mask**: patients with HD often have psychobehavioral changes (aggression, apathy, depression) years before the onset of motor symptoms
- 20. "Slow Park and Son": Parkinson Disease is a neurodegenerative disease commonly featuring bradykinesia (slow movement)
- 21. **Bill rubbing bribe**: a common feature of Parkinsonism is "pill-rolling" tremor (an involuntary circular movement of the fingers)
- 22. **Rigid safe cogwheel**: a common feature of Parkinsonism is "cogwheel" rigidity (ratchety pattern of resistance and relaxation on passive movement of extremities)
- 23. **Instable lifting posture** : a common feature of Parkinsonism is postural instability→ feeling unbalanced and increased tendency to fall
- 24. **Shuffling back to car**: a common feature of Parkinsonism is shuffling gait (due to bradykinesia and postural instability)
- 25. **Dangling double ropes**: first-generation antipsychotics (D2 dopamine antagonists) may causes symptoms of Parkinsonism
- 26. Accumulating copper pennies: accumulation of copper in the striatum (as well as direct binding of dopamine by free copper) can cause Parkinsonism in Wilson's disease (copper metabolism defect leading to cirrhosis)
- 27. **Dope-rope pulled out of black bag**: Parkinson disease is caused by a loss of dopaminergic neurons in the substantia nigra (pars compacta) → low dopamine levels
- 28. Pale mask by rope bag : dopaminergic neurons of the substantia nigra contain dark pigmented neuromelanin→ pale appearance of substantia nigra with loss of neurons in PD
- 29. Villainous pinSTRIpe robber with SUBmachine gun: loss of substantia nigra function → DISINHIBITION of striatum→ ACTIVATION of subthalamic nucleus→ INHIBITION of thalamus (decreasing motor function)
- 30. **Frozen thalamic switchboard operator** : substantia nigra dysfunction→ activation of the striatum→ activation of subthalamic nucleus→ INHIBITION of thalamus (resulting in motor dysfunction)
- 31. **Tazing submachine gun**: deep brain stimulation is sometimes used to inhibit the subthalamic nucleus and improve symptoms in PD